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THE

NATURE AND AFFINITIES

OF

TUBERCLE;

BEING

THE GULSTONIAN LECTURES FOR THE YEAR 1867.

BY

REGINALD SOUTHEY, M.D. Oxon.

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS;

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SIR THOMAS WATSON, BART., M.D., F.R.S.

LATE PRESIDENT OF

THE ROYAL COLLEGE OF PHYSICIANS,

THIS SMALL VOLUME

IS INSCRIBED,

AS A

MARK OF PROFESSIONAL ESTEEM AND PERSONAL REGARD,

BY

THE AUTHOR.

PREFACE.

THE Gulstonian Lectures, which I had the honour to deliver this year, and of which an abridgment has been already printed in the columns of the "British Medical Journal," are published by me in the present volume in their original entirety, at the request of several members of my profession who listened to them with favouring attention. They have been carefully revised, but the only real change to which they have been subjected is one of dress. They now appear divided into chapters instead of into lectures.

Two reasons have prompted me to make this alteration. Three lectures were somewhat narrow limits for the discussion of the subject which I had chosen, and I was therefore compelled to press all I could into each hour allotted to me: the halting points hence became much less a matter of choice than of necessity.

But when a lecturer publishes his ideas in book-form, his readers have a right to ask for better appointed breathing-spaces: this end is most conveniently attained by the division of his subject into chapters; a method, too, greatly facilitating the compilation of a table of contents, without which I reckon almost any printed volume seriously defective.

My selection of a strictly pathological subject is in accordance with the expressed wish of the founder of the lectureship, who directed the lecturer to give, so far as it was possible, a practical demonstration only of some process of disease upon the dead body.

In themselves these pages contain little that can at all fairly be entitled original research; indeed, the chapter which enumerates the separate anatomical features of the different forms of lymphatic tumours, and discusses the elements common to them all, is no more than a feeble review of a most able chapter upon this subject, contained in the valuable work of Professor Virchow upon morbid swellings.

My sole aim and whole endeavour has been to search for information upon the nature of Tubercle, to collect together the knowledge that has of late years been brought to bear on this most in-

teresting inquiry, and to put it into readable form, for the profit of my professional brethren, and, I will hope, for the benefit of suffering humanity.

It will be observed that I take little notice of the recent researches of M. Villemin upon the inoculability of Tubercle, published in the "Comptes Rendus," tome lxi., 1865, pp. 1012—1014.

Rabbits and Guinea-pigs, the animals usually selected as subjects for these experiments, present, even when they are improperly kept, or only badly fed, morbid appearances in their lungs, spleen, liver, and other glands, which somewhat closely resemble Tubercle of man; but, in common with Dr. Andrew Clark and other pathologists, I would urge that the identity of these two products of disease is any thing but proved; indeed, such microscopical evidence as we possess points to minute anatomical differences existing between them. Further, the fact of this animal abnormality being sometimes of spontaneous origin should make us forbear drawing too hasty inferences from its existence after an inoculation of the animal with human Tubercle: surely there is a probability of its nature being more akin to those metastatic, ill-nourished patches in man which have obtained the bad name of pyæmic deposits.

At all events, in the present pages I have deemed it wiser to avoid entering any further upon the field of inquiry thus newly upturned, and which to my mind does not offer much promise of future fertility.

32, MONTAGUE PLACE, W.C.,
Sept. 26, 1867.

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THE

NATURE AND AFFINITIES OF TUBERCLE.

CHAPTER I.

THE name of Tubercle was applied by the old medical authors to any little tumour. The root *tuber*, from which the diminutive *tuberculum* is derived, was used by Pliny when speaking of the fruit of a variety of apple: "Inter malorum genera recensetur: peregrina sunt zizypha et tuberes[1]." Pliny also used the word *tuberculum* in reference to tumours upon the scalp: "In capite multa variaque tubercula oriuntur, γάγγλια, μελικηρίδας, et ἀθερώματα nominant" (sc. Græci)[2].

Celsus again uses *tuberculum* as a translation for the Greek παρουλίς, a gum-boil: "Solent etiam interdum juxta dentes in gingivis tubercula quædam oriri dolentia, παρουλίδας Græci appellant."

The term Tubercle, thus originally vaguely applied upon a slight analogy of spherical shape to any little

[1] Pliny, i. 15, c. 14.

[2] Id. i. 22, c. 22, post med.

adventitious lump, has in the last hundred years obtained in modern European languages a significance so serious and important, that I have deemed it worth our while to refer to its earliest application in a medical sense, to learn, if I could, whether it had suffered from over or mis-use, whether the original little apple idea had been frittered away, or whether, rolling over the lips of so many generations of men, the term might not, like the snowball from the mountain peak, have acquired an expansion and *vis eundo* and vice by ill omen, which justified us in getting out of its way: but when I find Herr Virchow in his Cellular Pathology (p. 477) likening large conglomerate Tubercles to a Borsdorf apple, I receive the old analogy back again with a hearty welcome, perceiving that there must exist a similitude real and useful for purposes of illustration, and descriptively accurate enough to justify our adherence to a time-honoured word.

Now, the subject which I propose should occupy us in to-day's lecture is the real nature of Tubercle; but before we can discuss this we must agree to understand one and the same thing under this name. It is always interpreted, as Dr. Aitkens says, to refer to adventitious masses. The characters of these masses are, however, any thing but fixed: thus we have Tubercula Syphilitica, Mucous Tubercles, Tubercula Scrofulosa, and Tubercula Carcinomatosa, when the word simply implies an exuberant nodular growth; and again we find Tubercle bestowed as a

special name upon two very different kinds of things found in the body; the one being an irregular-shaped mass of cheesy or cretaceous consistence, and the other a pearly, grey-coloured, semi-translucent nodule which individually never attains any size; and lastly, we have a mixed substance, partly transparent, partly yellow and opaque, entirely adventitious, but confined to no particular form, and limited to no particular size, included under the same designation.

But if the term itself be a vague one, the views entertained upon the mode of origin of the material thus especially signified, are at least equally diverse: for one class of pathologists consider it to be an exudation, and speak of it always as a deposit, while others hold it to be a retrograde metamorphosis of pre-existing tissue elements, and a third more modern school, who receive their doctrines from a master teacher at Berlin, reckon it to be a new formation proceeding by direct growth out of a particular tissue. These last attribute to it a structure and development of its own, but a very limited capacity for organization.

There is yet a fourth class, the chemical pathologists, who entertain somewhat peculiar views of Tubercle, supposing it to fall as a precipitate from the lymph, while this is still within the lymphatic vessels, and in parts where lymph and blood are first exposed to increased oxidation[3].

[3] Simon's Lectures.

All these views will fall more conveniently under our notice hereafter; but I cannot help thinking that the three forms of Tubercle as at present almost universally accepted by pathologists, would, both for the sake of accuracy in definition, and for purposes of careful clinical observation, be far better dissociated; indeed the grounds for thus classifying them together under one head are, as I shall subsequently have occasion to show, insufficient; and the grouping appears to me justifiable only on account of the original vagueness conveyed in, and allowed by, the literal meaning of *Tuberculum*.

If all were agreed to attach to the expression no more sense than that of a little adventitious knob, I should certainly not quarrel with it; but custom and common use have restricted the application of the term within certain limits without fixing the bounds to these with any thing like sufficient precision. Tubercle and Phthisis are and have been daily used as if they were exchangeable words.

I cannot offer to substitute any other name for Tubercle that would have any chance of being accepted; Fuchs did so many years ago, when he proposed to apply *Phyma*, signifying the thing grown, to grey Tubercle. The invention of new names never does away with the misuse of old ones, until the reason for the alteration is clearly understood, and until the disadvantage of a change is felt to be outweighed by the clearer definition and more perfect knowledge of disease hence vouchsafed.

There are at least three forms of Tubercle which I must ask you closely to examine with me. I shall inquire, how they came to be reckoned together; what features they possess in common; and what points of difference they severally present.

First, then, how came they to be reckoned together? Let us hear Rokitansky[1]: "The collective term Tubercle," says he, "is made to embrace sundry formations which have nothing in common beyond their outward form." He then proceeds to show that even these outward forms are very dissimilar.

The grey, semi-transparent granulation of Laennec is a roundish, resistant, solid nodule, of millet-seed size; examined under a moderate magnifying power, it is observed to be branched out more or less, being lodged betwixt and between the elementary parts of the tissues in which it is found, and interpolating its substance among them, although adhering to them only by reason of an indwelling property of tenacity.

The metamorphosis Rokitansky at first allowed this form, was only that of simple cornification, but latterly I believe he accepted Laennec's opinion of its occasional central softening.

The second or croupofibrinous form occurs only as an irregularly rounded, branched mass, having every variety of size, attaining sometimes to considerable diameter, and appearing upon free surfaces as gibbo-stellate layers of very various thickness.

[1] Syd. Soc. Trans. vol. i. p. 292.

The metamorphoses he allows to this form are softening and cretification.

His third or albuminous form consisted of poppy-seed-sized glutinous granulations, which were scattered through the parenchyma of an organ, and were mixed, and miscible in all proportions, with a greyish sero-albuminous semi-gelatinous humour that infiltrated the diseased textures.

Rokitansky allows a subsequent cell genesis to all these forms, and minutely describes the cells thus developed *de novo*, the free nuclei, the "Punctmasse" free granules, free oil cells, and the molecular fat dottings of other observers; but he lays no particular stress upon any of these elements, as if neither the cells nor the nuclei presented any distinctive feature which could be relied upon for separating Tubercle from other things.

No; the argument, if argument it can be called, which he makes use of, comes to this: all these forms are exudations in the first instance, Tubercle in its broadest sense is an exudation, and therefore they are Tubercle.

An inquiry into the starting-point of Tubercle brings us, he says, always to a fixed blastema, which instead of organizing into a normal tissue abides at its primitive stage of crudity.

This blastema, he continues, exudes solid, by which of course he means that it solidifies close to the vessels out of which it comes, its nature being so highly coagulable that it cannot be deposited

far off its source of origin, inasmuch as its coagulation is due not so much to want of moisture (for it is often found in parts bathed by serous fluid) as to deficiency in vivifying influence from the surrounding tissues.

According to him all the original tubercular exudation does not form Tubercle; part of it is capable of reabsorption, a greater or less part of organization, and a rest only, or small portion incapable of absorption or organization, remains in the state of primitive crudity.

The doctrine of a primitive diversity in blastemata is the only tenable basis of a humoral pathology, as Rokitansky himself says, in the preface to his most valuable work, which is none the less valuable although it urges important fundamental principles with an allowed favoritism; and it is this diversity in the matrix blastemata which confers different endowments, according to him, upon the three varieties of Tubercle.

The three forms of Tubercle he gives us possess very little outward shape in common, neither does the point of similarity lie exactly in the blastemata in which they severally originate. These are by his own showing diverse; the likeness upon which he links them all together is one common basic substance, a qualitative anomaly of the fibrine of the blood. This is impressed with a tuberculous crasis, and every atom of tuberculo-dyscrasial fibrine becomes expended in the formation of Tubercle; and

it is the combination of this original element with other dyscrasial constituents that gives rise to the several different varieties of Tubercle.

Pursuing Rokitansky's own line of argument, one might answer that the dyscrasial constituents come, in the primitively yellow variety, to preponderate so greatly, and to alter the features of the adventitious product so completely, as to compel us to reckon this as an entirely different species.

The main objection, however, is that the latitude thus allowed for regarding any yellow inspissated pus mass as tuberculiform becomes a fertile source of error.

He found it necessary to account for appearances that were variously combined and grouped together in the bodies of persons who had died of pulmonary consumption. Solid nodules were discovered scattered about the body, and never at any distance from vascular structures—they must originate forsooth in blastemata which had solidified: they were organized in some degree, containing cells and nuclei—this organization, he concluded, must have been an after process, a development by Cytogenesis in the exudation. The one was more opaque than the other, because it possessed more "Punctmasse;" the one incorporated itself more into the tissues that surrounded it than the other did, it was because the blastema out of which it came was combined with a larger proportion of ordinary organizable fibrine. To the question, why does this exudation

thus immediately coagulate? he answers, it is its property to solidify. Where then is the original error? It is in the blood whose fibrine is dyscratic; and finally, we have the evidence upon which this assertion is made. "In the most acute tuberculous dyscrasia of the blood, the coagula found after death are soft and watery; they fully harmonize in their elementary composition with the Tubercle of exudation, that is, the Tubercle of the tissues[5]."

It is manifest that he classifies these three forms together under the common name of Tubercle, because, as he believes, they arise from one and the same blood dyscrasia[6].

Similarly Cytogenesis, or the development of cells *de novo* in an amorphous blastema, is a gratuitous assumption, and in the formation of grey Tubercle all the evidence we have tends strictly to its contradiction.

The small granular nodule vesicular Tubercle, as seen upon a serous membrane, is never found gelatinizing, or setting into a central solid form, while surrounded by an amorphous fluid. It is composed of structured elements from the very first moment that one can recognize it at all under the microscope.

[5] Lib. ante cit. p. 381.

[6] But this is neither argument nor logic; the whole question is simply begged: and with equal accuracy any disease or pathological degeneration that is attended by the formation of soft watery post-mortem blood-clots might be argued to be of tuberculous nature.

The normal histological cells of its immediate neighbourhood pass by successive phases of development and by transitional forms into the free nuclei massed together, constituting its middle and most characteristic ring, and these again gradually merge into the fine opaque central granules.

I shall next examine the doctrine inculcated by Lebert. Tubercle, according to him, is pathognomonically marked by Tubercle corpuscles, and these are his only ground for separating certain adventitious materials found in the body from the products of simple chronic inflammation. He attributes three forms to Tubercle, the grey, the yellow, and the infiltrative form: the common feature present in them all is his Tubercle cell; he terms them deposits (depôts), and considers that the immediate proximity of blood-vessels is indispensable to their origin.

He maintains that yellow Tubercle is a separate and distinct variety from its earliest existence; that although the grey passing through one of its phases of development can soften, smelt down, and become yellow, yet that the miliary yellow tubercle is a thing quite apart and by itself.

Both, he allows, may occur together at the same time and in the same part, as upon the pleural serous membrane.

The most characteristic grey type has its favourite site in the elastic tissue which supports the bronchi, the air vesicles, and the vessels in the lungs, and in the meshes between the elastic fibres themselves. He

is not, however, minutely precise here; for he confesses the impossibility of speaking positively to the site of the primary deposit, since the ultimate air cells and smaller bronchi all come to be involved in the miliary granulation.

He finds the most characteristic sample of the yellow variety in the lymphatic glands and in the submucous tissue of the intestine.

Subsequent and very careful investigations have however not merely deprived the Tubercle corpuscle of Glüge and Lebert of its specific value, but have gone far to show that from the moment cell forms of the kind he describes appear in any deposit, the structures really characteristic of Tubercle are in process of disintegration.

His typical cells, moreover, are in their early state indistinguishable from lymph cells, as these are to be seen in healthy glands; and in their blighted, shrivelled, degenerated condition are no way dissimilar to pus cells in inspissating pus.

But more than this, his reason for thus uniting the three forms of Tubercle together is not merely insufficient, but leads directly into error; for the little grey granulation of tubercular meningitis, the most typical example of Tubercle that we know of, in its early state presents no such Tubercle corpuscle, and becomes excluded from his category altogether; while many forms of chronic abscess, which do not in their mode of origin, or nature, or attributes possess any thing in common with Tubercle,

might thus with equal propriety be included under this head.

Hence arose that widely-spread, yet most fallacious notion, that Tubercle and caseous degeneration were synonymous terms.

The latest and most definite views of Tubercle that we possess have been recently published by the author of the "Cellular Pathology," and these it behoves us to consider next.

Tubercle, according to Virchow, is a new growth, and is classed by him among the Lymph tumours, those namely which are constructed after the pattern of lymph glands, and which stand most closely in relation to connective tissue formations[7]. The single tuberculum or the tuberculous tumour is not capable of identification from any one element entering into its composition; but its origin, its development, and its minute structure together confer a particular stamp upon it, as a whole, which renders it capable of distinct recognition.

It will be necessary for me to describe the minute anatomy of Tubercle, as this is to be generally gathered from Professor Virchow's writings, and not a little from his teachings; for I had the privilege of learning much from him personally some few years ago.

The Tubercle formation, he shows, is cell-structured from the moment of its first appearance; it

[7] Die Krankhaften Geschwülste, Band ii. S. 557.

proceeds always out of connective tissue, or from some tissue closely allied to this, such as false membrane, fat, or the medullary tissue of bone. It exists in two forms, the one he terms the cellular, the other the fibrous form; but they have such features in common as imply unmistakable oneness. The fibrous form is only a slight structural modification of the simple cellular—a modification, as I shall hereafter have occasion to show, impressed upon it by the external conditions of its growth.

The origin and mode of development of the simple cellular form is best of all to be studied from the tuberculous growth as this is found upon serous membranes, or upon the mucous membrane of the larynx, and after these in organs like the liver and kidney, which are endowed with a distinct stroma of their own. Virchow's description[8] is from an example upon a serous membrane. The young growth is somewhat smaller than the smallest millet-seed; it has a granular look, and contains soft, imperfectly developed cells, which are very easily broken down, and free nuclei. Its elements, although differently grouped, are identical with those that constitute a normal lymph-gland.

The isolated Tubercle forms the tiniest tumour that occurs on the human body, but it is rarely if ever single. These growths are almost always multiple; they are found in nest-like groups close

[8] Lib. ante cit. p. 636.

together, and multitudes of nodules thus originally and individually distinct can combine together and form a conglomerate tumour.

Conglomerate Tubercle increases in size by surface accretion; new nodules grow up in the tissue immediately round about the old ones; and as this accretion can take place from all sides around a centre in the parenchyma of solid organs, the final shape attained by the mass is round or roundish.

But upon free surfaces the extension must follow the plane of the matrix tissue, and hence the form ultimately attained is more or less flat or squab. The composition of Tubercle is cellular wherever the growth can pursue its natural course unobstructed.

In studying the features of a new growth, just as in the examination of any tissue, there is a point which must be borne in mind, namely, that the structures of which it is composed will present themselves in different stages of development.

1st. There are the young embryonal elements, the cells which may be called indeterminate, such for example as those of connective tissue in the corium proper, the deeper layers of the cutis.

2ndly. There are the determined or special cell and intercellular developments, which give by their shape or arrangement its particular stamp or seal to the tissues to which they belong, as bone cells to bone, cartilage cells to cartilage, and Tubercle cells to Tubercle.

3rdly. There are the forms presented by the structures in process of disintegration towards removal *e situ*—such, for example, as are the dried, broken, granularized upper scales of effete epidermis, the fatty degenerate mucus cells from mucous surfaces, the central fine granules of tubercular débris.

Now the pathognomonic features of a new growth, like the characteristic elements of any normal tissue, must be especially read off and estimated at the second stage, when the component parts have attained to their highest state of perfection. This is a sort of pause period, which is of very different duration in different tissues. It is at this pause that those structural features predominate which enable us to form an opinion upon its nature: it must be further remembered that the elements of greatest permanency in an adventitious product do not by any means necessarily correspond with the structures of highest aim in it.

It is true that the distinguishing elements of a healthy tissue are not only present in greatest abundance, but are themselves most permanent at the period when they are most characteristic.

But almost the very converse of this might be announced of abnormal growths. Their stage of greatest permanency is invariably somewhat removed from that of their highest perfection—they incline always to continue longest either in their embryonal or in their degenerative stage. Tubercle, as an instance in point, can be affirmed to do both;

since in its cellular form the tendency is to pass most rapidly towards, and to remain most permanently at, the degenerative stage, and in its fibrous form at the embryonal.

In studying the histology of the isolated grey nodule, which is the most honest sample and purest specimen of Tubercle, the three ages of development above described are all most distinctly visible. Outermost lie the connective tissue cells in process of endogenous proliferation[9]; next come the highest aimed structures which the growth reaches—the cell forms most characteristic of it, which are disposed in an irregular ring round the centre, and which, doubtless, multiply themselves by fissiparous development; and lastly, placed most centrally in the examples offered by Tubercle in the stroma of solid organs, granular amorphous bodies can be seen which might not be inaptly termed the products of the eremacausis or smouldering combustion of animal tissues. Commensurately with the age of the growth, these fine granules and fatty compounds, significant of the stage of decay, encroach upon and predominate over the cellular structures.

The Tubercle cell, says Wedl[1] (and this is not that cell on which Glüge and Lebert laid such stress—that was a compound granular corpuscle, an element of much later formation in the stage

[9] That is, multiplying by producing new cells in the interior of old ones.

[1] Pathol. Histol. pp. 367, 388.

of caseous metamorphosis), is a real cell, not a nucleus or a solid body; it is usually smaller than a white blood cell, but it can attain to double or treble this size; it is round, like the leukæmic typhous or scrofulous cell, and belongs to the nature of a lymph-gland element; it is colourless, transparent, and faintly granular; when fully developed a shining nucleus is to be seen in its interior, a nucleus which in some instances is small and homogeneous, in others more granular and nucleolated: a few of these cells have two or more nuclei.

Although the outer layer of the middle ring is thus indisputably composed of perfect cells, between which a fine reticulated meshwork of connective tissue fibrillations extends, still by gradual transition a layer of what cannot be distinguished from free nuclei is quickly reached, and these are packed so close together as to admit the interposition of no interstitial substance whatsoever: further, the transition from these again to the amorphous central granules is through a series of gradually diminishing nuclear forms.

The multiplication of nuclei peculiar to the outer layer of connective tissue is, says Virchow[2], to be seen to greatest advantage in the tubercular growths which proceed out of the fat-cells of the omentum. Here the fat is first absorbed, the nuclei in the interior of the cells divide by repeated fission, they multiply so as to distend the cells to gigantic size,

[2] Virchow's Archiv, Bd. xiv. S. 49.

and are pressed so close together that it is easy to imagine one is looking at nothing but free nuclei.

Now, if the nature of the tuberculous growth had to be decided upon the intrinsic value of some one of its component elements, it would be to these angular-shaped free nuclei thus herded together that I should attribute paramount importance.

Its cells, although these are heterologous, wrongly developed (a term whose full value I shall hereafter have occasion to discuss), do not really serve to distinguish it from other things; neither does its peculiarity depend upon the proclivity of these pitiful cells to degenerate, nor attach to the free nuclei or central granules. The truth is, that the whole affair is quite out of order in the place where it is found; none of its structures considered separately suffice for its identification; but, occurring together upon a type which is constantly repeating itself, they are eminently significant.

Lymphous elements proceeding out of connective tissue are no anomaly, but lymph-like cells arising out of connective tissue by endogenous multiplication, and showing no further capacity of developing intercellular substance about themselves, coming to press against each other so as to impair each other's shape, and interfere with each other's nutrition, quickly reproducing mere nuclei, which pass no higher, but pulverize again into amorphous matter. This entire group of changes I apprehend to be not only peculiar but pathognomonic. Developed by

a wrong method, and in a part which ought to present nothing of its kind, Tubercle is a new growth capable of being recognized by its characteristic build. It does not develope *de novo* out of a blastema; it is organized from the first moment that we are able to recognize it, although essentially incapable of high organization.

When a caseous or cretaceous mass is presented for our examination, we are justified in deciding upon its pathological nature only from our knowledge of its mode of origin. If there be no appearances in or about it which correspond with the structures above described, we have no right to call it Tubercle, and we shall act wisely if with Professor Virchow we restrict the application of this term to the material he has thus closely and minutely described.

The single cellular tuberculum is rarely much larger than a pea, but this which may perhaps be taken as its extremest size is occasionally reached in the mucous membrane of the intestines, and in the fibrous sheath of the gall-ducts. In the stroma of the liver and kidney I have often alighted upon Tubercles which were so small as to be only microscopically visible.

I shall next consider the form and shape attained by the tuberculous growth in the progress of its further development; for it remains to be shown, what Carswell first propounded, how much this is affected by external influences.

Tubercle in parts like thick false membrane, or

firm fibrous tissue, does not attain its most typical shape, or degenerate quite in its ordinary way. The eruption of small-pox is a very different affair upon a mucous membrane from what it is upon the skin: occurring in a firm fibrous tissue, and removed a certain distance from vascular supplies, it partakes in some degree of the nature of its matrix, and is proportionately disinclined to soften and break down.

Virchow designates this the fibrous form; it is the infiltration form of the older writers, who were not slow to recognize it as Tubercle by reason of more typical appearances in its immediate neighbourhood. Its development is in this wise: the connective tissue cells are impressed with doubly wrong propensities; while certain of them give birth to a degenerate brood of tubercle elements, certain others pursue a less vicious bent; the nuclei divide again and again, but attain a certain fibrillation so as to produce an ordinary hyperplastic increase of substance. The two changes may complicate each other in every possible degree, but it is found that the further off blood-vessels heteroplasia[3] and hyperplasia[4] are being conducted, the more the hyperplastic aim predominates: in this way an enormously thickened mass can be produced whose surface is perfectly smooth.

[3] Heteroplasia means increase by development of new cell elements dissimilar to those proper to the part.

[4] Hyperplasia signifies increase of size by multiplication of normally pre-existing cell elements.

Rokitansky very conveniently attributed this condition of things to an origin of the substance out of mixed blastemata, saying that the tuberculous exudation was capable of mixture with simple fibrinous or croupo-fibrinous lymph in all proportions; closer investigation, however, interprets the affair not merely more reasonably, but more accurately, in accordance with the doctrines of a cellular pathology.

In the formation of false membranes, as in pleurisy, for example, the organization begins in contact with previously organized parts; a clear fluid may exude from congested capillaries into the cavity of the pleura, but the cell proliferation, by means of which new tissue actually is formed, proceeds from one or other serous surface. A section of the thickened pleura demonstrates these cells in several layers of progressive growth, the embryonal forms lying deepest. Now, in several places these cells remain sufficiently long *in situ* to flatten, lengthen out, and adhere or fuse together at their edges so as to form a permanent tissue. But every where this is not so: a large portion of the growing cells bud out as it were into the fluid by which they are bathed—itself an unfavourable item towards their persistence—and are shed off in this their embryonal state. Here then is development into an exuded fluid, not out of it; and the assumption of these free floating cells having originated *de novo* in the fluid is gratuitous, incapable of proof, and unlikely, because con-

trary to our experience of development in other parts.

I recently had occasion to examine an enormously thickened tuberculous mesentery coming from an adult, who had died of acute tuberculosis; the appearances were such as I think could only be explained upon the hypothesis of intermingled growths.

The surface of the mass was perfectly smooth, neither nodular nor wart-like, so that one might fairly have questioned the existence of Tubercle in it at all, if our conception of this had been derived from the idea of knobs or knots, and tubercle corpuscles, such as Lebert described, were not forthcoming in it. Upon section, irregular whorls or upheavings of some transparent pearl-like substance, set in a much more opaque felt-like material, could be seen by the naked eye. Beneath the microscope the more transparent parts were observed to be composed of connective tissue cells in process of ordinary hyperplasia, clear nuclei multiplying every where, and being every where round and distinct; but in the opaquer parts were situated larger and more irregularly outlined nuclei which had a granular look, and were mixed in all proportions with amorphous matter. These last were the Tubercular Heteroplasias; here were the elements of Tubercle—nuclei in process of rapid disintegration into molecular forms. But the manner in which the growths were dovetailed into each other, like the compound papillæ of the skin in the mucous layers of the epidermis, showing

groups of distinct elements united together by transitional forms, left no other method, except that of co-development and common source of origin, to explain their interweavings. One might as reasonably pretend of a linen sheeting, in which the regular in and out cross-thread marked the passage of the shuttle, that it had been produced as metal gates are formed in the piece at one casting.

CHAPTER II.

THE ORDINARY COURSE OF THE TUBERCULAR GROWTH.

THE elements of Tubercle are eminently short-lived and prone to degenerate, and the natural course of this degeneration is a fat metamorphosis. It is a moot question if Tubercle can ever be reabsorbed without passing through this retrograde step. Virchow speaks to the possibility of such complete resolution taking place[1], and there are some few clinical observations rendering it not improbable. The fat metamorphosis of the tubercle cell differs in many respects from ordinary fat degeneration. This change in a renal epithelium or catarrhal mucous cell may be taken as the type of the usual process, according to which the fat granules accumulate in the cell, first distend it, and then run together into one or more large fat drops in its interior; but in the tubercle growth from the very commencement of the change the cells diminish in size, shrivelling from loss of fluid contents, while the fat which dots both cells and nuclei with fine opaque granules presents no tendency to run into oil-globules.

[1] Würzburger Verhandl., Bd. vi. S. 11.

It is this feature of a retrogressive metamorphosis, conducted under conditions of peculiar deficiency in moisture, which affixes a special stamp to the subsequent proceedings.

Thus the simplest issue of the most uncomplicated form of Tubercle is a species of dry crumble from within outwards. The denser, tougher, and more fibrous the structure is in which Tubercle is developed, the more slowly this retrogressive change is brought about, whereas the softer and moister the matrix tissue of the growth, the more rapidly this breaks down and shells out. An intermediate state of its surroundings in which these are neither especially moist nor dry, of course the most common condition, favours the transition of Tubercle into a cheesy mass and its persistence in this stage. Finally, grey and yellow Tubercle differ from each other only in the amount of accidental fat elements they respectively contain, the more yellow Tubercle being the one naturally richer in fat.

The miliary nodule seated in the parenchyma of a solid organ tends to soften in its centre; the grey granular Tubercle seated upon a free surface softens at a peak point; peripheral softening affects only the conglomerate form; and the change then ensues, not spontaneously from any innate propensity in the mass itself, but from external influences, such as inflammation or any excess of moisture in the neighbouring tissues will produce.

I shall proceed next to consider the progress and

changes which the tubercle growth exhibits when subjected to different external conditions: its ordinary course is, as we have just noticed, towards degeneration, and this with greater or less rapidity, as the development of the new growth proceeds under exposure to great or little moisture.

TUBERCLE PRODUCING ULCERATION.

First, when exposed to great humidity.

When superficially situated upon a mucous membrane, as in the larynx, Tubercle presents us an example of its most rapid degeneration. The growth here takes place subject to conditions of greatest external moisture, the caseous state is then not reached, and the whole softens and shells out, leaving an ulcer, which from the rapidity of its formation, and non-cheesy nature, was for a long time supposed not to be of tuberculous origin. Louis entertained the idea that these ulcers were no more than excoriations produced by the acridity of the phthisical sputum [2]. Virchow recommends them, however, as very typical instances of the progress of the growth [3]. The ulcer which has thus begun may extend into the submucous tissue, and its walls then can become infiltrated with characteristic cheesy material, but in the larynx these sores rarely attain to a size larger than that of an ordinary split pea.

[2] Louis, Recherches Anat., Path. et Ther. sur la Phthisis. Paris, 1843, p. 51.

[3] Virchow, Krankhafte Geschwülste, Bd. ii. S. 645.

The further course of such tubercular ulcers is best studied upon the mucous membrane of the bladder; they spread at their edges and in their floors, the submucous tissue thickening enormously beneath them, and becoming the seat of crop after crop of new growths. At almost any period of their existence the walls can soften a little further, clean themselves by secretion of laudable pus, heal up and scar over, as is often seen in the intestines, where the ensuing constriction is not always the most favourable event, but for the most part the sores extend one into the other, forming irregularly branched rodent ulcers with thick overhanging borders. Malignant exactly they are not, but there can be no question of their infectious nature—the disease spreads by propinquity (juxtaposition), and the scar tissue of an old tuberculous ulcer is the favourite place for new nodules to form in.

The only two tissues that appear able to resist actual disintegration among the débris of tubercular ulcers are bone and elastic tissue: these, although dead and separated, for long retain their characteristic microscopical features unchanged.

Although at first calculated to surprise us, perhaps the second best illustration of the course of Tubercle under conditions of greatest moisture is to be found in bone tissue. The favourite nidus of Tubercle in bone is the medullary substance, and the disease therefore occurs especially in the spongy bones, and in their spongy cancellous parts, thus principally

affecting the vertebræ, the articular ends of the long bones, the tarsus and carpus, and the skull-bones of children. At its late as at its early stages, the tubercular nature of the affection is difficult of recognition, for large cretaceous masses are never formed—the grey granulations usually pass into ulceration before they can be observed; and macerating the bone, the method of investigation generally adopted brings out the ultimate feature of the caries, the bubble-like perforation, into bold relief, but completely obliterates the structural changes most characteristic of the disease. And further, those tunnellings and thorny persistent bony ridges, so aptly designated by the name of Teredon or Spina Ventosa, are appearances pretty nearly as often attained by scrofulous or other ulceration of bone as by the tubercular process.

There is, however, a significant clinical symptom which may serve to distinguish the tuberculous disease of bone, namely, the relative absence of periosteal thickening about the painful part. The bone itself becomes swollen and tender long before its periosteum sympathizes with it.

"Necrosis chronica sive occulta" was a very sound definition of this affection, given to it by Ter Borch [1]. The admirable description of the disease by Corn. Black assists us greatly towards understanding the manner in which the tubercle growth is developed, not out of an exudation, but in the substance of a tissue.

[1] F. G. Ter Borch, Verhandelung over de Necrosis. Amsterdam, 1821, p. 104.

The medullary cavity is every where filled with a vascular membrane. This is a fine areolar tissue which contains the blood-vessels and extends through the Haversian canals, passing by direct continuity of structure into the fibrous matrix of the bone. In its state of highest perfection this membrane presents, as he (C. Black) says, "a basement structure, and is surmounted by a layer of flattened, irregular shaped cells." Now [5], cancellous or spongy, bone differs only from the compact by the predominance in it of soft interstructures relatively to the consolidated. The bones of the fœtus do not possess the perfected medullary tissue of the adult; they are filled with a transparent reddish fluid which is only a little more tenacious than bloody serum; and the younger a bone is, the softer and more embryonal is this vascular areolar lining tissue.

It is found that in the bones of young subjects Tubercle can develope itself in the soft and medullary substance without this undergoing any previous change; but in adults, when the tissue is firmer and more consolidated, a stage of active congestion and softening, a step backwards towards the embryonal state, precedes the miliary formation, a point of interest which I cannot forbear mentioning.

The Tubercles, as Nélaton [6] describes them, are

[5] Compare Quain and Sharpley, vol. i. p. cxxiii.

[6] A. Nélaton, Recherches sur l'affection tuberculeuse des os. Paris, 1837, p. 22.

first met with as small, clear, grey, scattered granules, which a little later may be grouped together; they degenerate quickly into an opaque yellow material, with which the products of ordinary inflammation are combined in all proportions. The walls of the cancelli, the vascular membrane, the cavities, lacunæ, and canaliculi become stuffed with cells, free nuclei, granular matter, and oil globules of various sizes, the larger ones sometimes completely filling a lacuna.

The circulation is greatly impeded, and in parts completely arrested, the blood then flowing in extra quantity through other channels, so that in one part degeneration and absorption of tissue can be taking place, while in another, growth of osseous tissue or exostosis from the walls of the cancelli through an increased supply of nutritive materials, and thus it is that secondary sclerosis takes place around the sites of the tuberculous growths.

If the crops of primary growths are numerous, and the conditions of surrounding softness and vascularity such as principally favour their early degeneration, as obtains in the bones of young children, which are nearly altogether composed of spongy tissue, the whole may be speedily converted into an ill-conditioned pus-like fluid. Virchow has given an admirable description of a phalange being thus entirely destroyed.

But with adults and in more completely formed bones the compact substance as above described

gets re-converted into a soft granulation tissue in which the miliary growths develope, and it is this anticipatory red swelling of the bone which confers an appearance of encapsulation of the tubercle crops described by Nélaton[7], itself probably no distinctive feature of the tuberculous process. The disease may ultimately lead to the necrosis and separation of small portions of spongy bone tissue through fistulous passages, and when the external air has once gained access, secondary simple bone abscess, with all its attendant phenomena, may ensue. The articular cartilages are never, so far as I know, implicated in the tuberculous process, but may be hypertrophied if in close proximity to the new growth; so too they may secondarily ulcerate through the interposition of a false membrane between the synovia and the cartilage, the supply of nutritive fluids being then intercepted.

As a rule, the disease in bone is one in which the new growth stage is of very short duration; caseous material is less frequently formed by this than by the scrofulous process: indeed it may be accepted, upon Professor Virchow's assertion[8], that the large cheesy accumulations about diseased vertebræ are scarcely ever of tuberculous origin, but are the result of Osteo-Myelitis Scrofulosa. As to the resolution and prognosis of the two affections, this much can be

[7] Nélaton, l. a. c., p. 15.

[8] Lib. ante cit., p. 712.

vouchsafed upon the same authority, that the scrofulous disease is capable of complete self-cure; but that, although the tubercular bone ulcer may possibly heal, the loss of substance it has caused never is repaired.

TUBERCLE IN NERVE SUBSTANCE.

Tubercle in nerve substance presents us a very good example of this growth's development under conditions of neither excessive moisture nor dryness, when the tendency is for it to become a caseous mass. Since its matrix tissue is the neuroglia (the interstitial connective tissue of nerve substance), it may begin almost any where and extend in every direction: its site of predilection, however, is certainly the grey substance, whether of the brain or of the spinal cord.

When Tubercle occurs in the cerebellum, as it often does, it is sometimes difficult to distinguish the young tubercle cells from the nuclei of the large ganglion cells of this part; and again, at a later stage of their existence, the growths which are apt to be superficially situated and to attain to considerable size are liable to be confounded with syphilitic gummatous tumours. The features which serve for their interdistinction are as follows: the gummy tumour in the brain possesses what its name implies, a gelatinous consistence; it is uneven in shape, and from all sides passes by gradual transition into the surrounding brain substance.

The conglomerate brain Tubercle presents a round, smooth surface, and is encapsulated by a highly vascular false membrane of close connective tissue, which is the matrix tissue of the successive growths.

It is a matter of no little interest that Tubercle is most apt to form in the human brain in infancy and early childhood, that is, at that period of life during which we know this organ undergoes its most rapid nutritional interchanges, and therefore when a development of the neuroglia in a wrong direction need least of all surprise us.

Whether the primary miliary granules begin in the connective tissue of the brain or in the sheath of the smaller arteries is quite uncertain; for the commencement of the growth in these parts is still involved in complete uncertainty.

The earliest trace observed is a little caseous mass surrounded by a capsule; in this cyst wall crops of young nodules are found, which in the course of their growth run into each other and degenerate, forming a layer of substance which closely enwraps the central mass.

With the death of each zone of new growths, the capsular wall pushes out fresh nodules, which are thus continuously laid in contact with the former layers, and adhere to them: hence a section of these formations shows them to consist of several lamellæ.

The mass increases by a process of very slow and possibly of very irregular development, at one time, and under particular conditions of the general health

of the subject, pushing forward and making rapid progress, and at another remaining dormant, encysted and unirritating.

The length of time that conglomerate Tubercle of the brain remains in a caseous state, the size it reaches, and its presence among such important structures unattended by serious symptoms, form its principal characteristics.

Such caseous and cretaceous masses are often lighted upon in the brain in the course of post-mortem examinations, when their existence has been least of all anticipated; but, on the other hand, they occasionally excite the gravest possible symptoms during life. They sometimes break down into a fatty pulp, and produce active inflammation in the central substance, or else give rise to an extensive meningitis attended by numerous crops of secondary growths upon the pia mater, and in the walls of the smaller blood-vessels: confirmatory evidence of their primary and true tuberculous character is thus sometimes obtained at long posterior dates. The calcification and encapsulation of Tubercle tumours in the brain, that is, their reduction to a condition of complete dormancy, or self-cure, is no uncommon thing; but since cysticerci are often thus included, since simple scrofulous abscess may thus become metamorphosed, we should not hastily jump to the conclusion that every calcified adventitious product we alight upon in cerebral substance has had its origin in a true Tubercle growth.

TUBERCLE IN THE KIDNEY.

The last example I shall adduce, one illustrative of the progress of Tubercle in the parenchyma of a solid organ, is from the kidney. I know of no instance in which the constituent elements remain longer in their cellular stage, except of course that mixed growth of Tubercle with hyperplastic fibrous tissue occasionally found in the substance of a serous membrane, as in the tuberculous mesentery, mention of which has already been made: the external conditions here do not favour a so rapid change into the caseous state, and we are hence enabled to watch the successive steps of the process of degeneration more closely.

The kidney presents to the naked eye minute, white, semi-translucent dottings scattered throughout its substance. These are found to consist of multiplied nuclei massed together, and can sometimes be traced to direct proliferating sources in the connective tissue.

Spreading from numerous centres, the growths extend through the interstitial tissue of the organ, separating the tubules and capillaries more and more from each other. The nutrition of the renal cells within the tubes is thus interfered with, and these degenerate into fine granules: the blood ceases to circulate through the capillaries of the part, but permeates by collateral flux through other channels; shortly the outline of tubules and vessels

involved in the new growth becomes less and less distinguishable, until at last these highly organized parts are converted into a finely granular material, such as entirely corresponds with the ordinary central masses of miliary growths.

Finally, the scattered nodules, which looked for a time so like little fibrous tumours, extend into each other, their centres becoming yellow and opaque, until a caseous conglomerate is formed, which has nothing in it significant of the Tubercle growth, and might from its aspect well be esteemed a simple abscess.

This, then, may be taken as a fair example of Tubercle when developed under particular conditions of approximate external dryness, in a firm, as opposed to a loose, textured tissue, and the same growth in the substance of the liver affords us almost identical appearances [9].

A discussion of the various opinions which have been entertained upon the origin of Tubercle would, I fear, be labour in vain, a long and a profitless task; since the idea of a caseous mass and Tubercle being terms capable of mutual substitution stands in the way, and vitiates the premise and nullifies the deduction of the arguments in almost every instance.

Again, almost all the older notions of Tubercle

[9] To give a more detailed account of the pathology of Tubercle would involve our following its manifestations wherever it occurs in the body; which is unnecessary for the object I have in view, and would carry me beyond the limits to which I wish to confine myself.

have been derived from the study of lung affections, and although I am far from asserting that Tubercle is a rare thing in these parts, yet I must agree with Virchow that it is always difficult of recognition in pulmonary tissue; that it occurs in a typical state only in the walls of the smaller bronchi before these enter the alveoli in the interstitial, interlobular, or interlobar structures; and that the conditions of its growth in this organ are just those which principally favour its speedy metamorphosis.

They are reasons, not against the existence of Tubercle in the lungs, but against the foundation of our opinions as to its nature and origin too exclusively from its presence in them.

Further, under the name of phthisis or pulmonary consumption, a term of wide embrace, there are at present comprehended a multitude of morbid lung states, many of which are doubtless due to the ravages of tubercular disease, but some of which owe their origin to chronic pneumonia, while others are indistinguishable from simple abscess in a particular tissue, and appertain much more strictly to the scrofulous than to the tuberculous habit of body. M. Colin, in an admirable clinical memoir upon some cases of acute tuberculosis, speaks distinctly to the same effect, saying, "Faudra-t-il céder à cette pensée instinctive d'affinité qui nous fait toujours unir les deux mots Tubercule et Poumon[1]?"

[1] M. Colin, Mémoire Clinical, 1865, p. 71.

CHAPTER III.

It is worth our while briefly to examine how the different opinions of the humoral school of pathologists upon the nature of Tubercle have become indoctrinated, but more than this I dare not attempt.

Sylvius de la Boe, in 1695, stated that Tubercle arose from certain glands in the lungs which resembled those of the neck and mesentery; and out of this statement sprang the lymphatic doctrine of Tubercle as it was entertained by White, Barnes, Hufeland, Schultz, Andral, and several others. The explanation, if such it can be called at all, is to all intents and purposes the same as that still offered by the humoralists of the present day; and strangely enough it is the one which *mutatis mutandis* is just now presented to us by the cellular pathologists. For we find Dr. Rindfleisch of Zurich, in 1862, regarding Tubercle as a lymphatic gland imperfectly developed, and Professor Virchow, in 1865, testifying to the lymphoid character of the tuberculous tumour.

Carswell announced that he had detected tuberculous matter in the blood, and concluded that it was simply deposited from this fluid in the tissues.

Andral, in examining the body of a person who had died of acute tuberculosis, found the centre of a blood clot in the aorta studded with small round granulations like miliary Tubercle. White blood cells and tubercle corpuscles were constantly being confounded together as one and the same thing. Addison believed the white blood cells were extravasated and became themselves the tubercle corpuscles, and thus the lymphatic doctrine of Tubercle became merely the accepted recognition of a lympho-tuberculous blood dyscrasia.

The late Dr. Todd of Brighton thought that in tuberculosis an unorganized albumen, an imperfectly elaborated plasma, circulated with the blood and was deposited in the tissues; an idea further worked out by Dr. C. J. B. Williams, who taught that cacoplastic cirrhosed tissue formative lymph and aplastic tubercular lymph were varieties of ordinary coagulable lymph, from which too they differed not in kind but in degree of vitality and capacity of organization.

In tuberculosis, says Mr. Simon, the blood dies stillborn in the lymph stage. "The lymph is malformed, it is oxidized and falls down as a precipitate in the blood wherever this comes into contact with the air,"—an hypothesis evidently propounded to meet two acknowledged facts: 1st, the circumstance of the lungs being the commonest site of caseous deposits; and 2ndly, the immunity from tubercular disease afforded by cyanosis, or any cause that induces extra venosity of the blood. But how

does this dyscratic blood theory of Rokitansky, Henle, Simon, and the humoralists generally, almost invariably end? Why, by premising capillary obstruction by the means of an aplastic fibriniform material introduced into the circulation, and by deriving Tubercle directly from this capillary embolism.

Quekett and Rainey advanced an observation in support of the same idea, by finding degeneration of the coats of the arteries in people who had died of tuberculous disease, and strengthened their opinion upon the discovery that the blood vessels of rabbits which had been rendered artificially tuberculous (?) offered surprising obstruction to the entrance of injection fluids. Henle, discussing the origin of Tubercle in the lungs, considered that the first wrong step was a defect in the capillary circulation, a coagulation of the blood in the vessels.

Lebert and Peacock had noticed in several cases that aneurismal dilatations of the arteries accompanied tubercular developments, Peacock in the branches of the pulmonary arteries, and Lebert in the smaller vessels supplying the brain.

Dr. Cotton recently communicated to the public the notice of aneurisms of large branches of the pulmonary artery, and showed them to be not very infrequent sources of fatal hæmorrhages in the third stage of phthisis[1]. Bouchut observed the presence

[1] The explanation Dr. Cotton offers of the pathology of these aneurisms is certainly ingenious, and probably correct. He supposes that the walls bulge out in the first instance into cavities by

of granulations, not merely in the substance of the pia mater, but especially along the walls of the larger vessels of the brain, in children who had died of tubercular meningitis.

A more exact account of this change which the walls of the blood-vessels undergo, with an interpretation of its strictly tuberculous nature, was published by Dr. Rindfleisch in 1862. He found that the walls of the vessels were not only studded up to their finest twigs with grey Tubercle, but that the developments in question were still visible as spindle-shaped varicosities upon the capillaries themselves. His investigations led him to attribute the first step of the Tubercle growth to a hyperplastic increase in the finely granular substance of the middle coat of the vessels between the muscular sheath and the adventitia; accompanying this increase he observed the nuclei multiplied and became more spherical, the structureless adventitia being bulged out before them. He then thought that the protoplasm thickened into a cell wall around the nuclei, and that in this way the large Tubercle cells were formed.

According to His, however, there is a natural peculiarity in the adventitia of the vessels of the brain, and Virchow also allows them normally to

lack of their natural support on the side of the vomica; that the same cause which has led to the removal of supporting tissue also interferes with the proper nutrition of the arterial walls, and leads directly to their degeneracy.

possess large round cells, such as are not found in the connective tissue sheath of other vessels. Without then denying the likelihood of the origin of Tubercle in some instances from an actual misdevelopment of the arterial walls, we must remember that there exists abundant evidence in favour of its more frequent departure from their connective sheaths.

And further than this, the purely vegetative functions of the blood-vessels in the body militate strongly against the probability of their substance becoming primarily the seat of a new growth. My own belief—and this is formed upon careful examination of the blood-vessels, the smaller arteries of the choroid plexus in the ventricles of the brain, in several cases of tubercular meningitis—is that the new growth takes origin in the adventitia or external connective tissue sheath; that this becomes visibly thickened, bulged, and more than naturally opaque, so as to be easily discernible with the naked eye; and that the muscular coats are secondarily involved as the little tumour extends in miniature egg shape, both into the substance of the wall of the vessel, and also outside it, perhaps intruding itself into the surrounding parts. An examination, with a high microscopic power, of this opaquer than natural spot in the wall of the artery awakens us at once to the fact, that the abnormality consists entirely of a nuclear proliferation.

Now I should not for one moment dispute the fact that tuberculosis is preceded by a distinct blood error, that the disease may with a show of justice be argued to start from some mal-assimilation of the food, and possibly to spread, as Dittrich taught, by a re-absorption of the products of proteine metamorphosis into the circulating fluid—indeed one must allow of capillary obstruction as the common consequence of the progress of the disease: but the question at issue still remains just where it was, we shall not have advanced our knowledge of the pathological process one "Iota" by these speculations, and must inevitably return to the first and only actual evidence of definite error—the Tubercle growth which as yet has only been distinctly traced developing itself out of the connective or some correlative tissue.

The evidence of Schrœder van der Kolk, admirable although he is in all pathological research, cannot, to use a legal phrase, be brought into court here: he deduced his opinion, just as Henle and Reinhardt did theirs, from the catarrhal multiplication of cells that takes place by endogenous development out of the epithelium lining the ultimate air-sacs of the lungs.

He found the pulmonary vesicles distended with swollen spherical cells: the smaller ones lay against the walls; the larger, which contained several nuclei, and were filled with granular matter and oil-

globules, quite like compound granular corpuscles, lying centrally in the cavity of the air cells or in the bronchi.

Now this is exactly converse to the arrangement of the elements in the Tubercle growths, the centres of which are granular, the outer circles of them only containing cell forms; and these appearances are themselves quite indistinguishable, so far as I am aware, from grey hepatization of the lungs.

CHAPTER IV.

Two terms were made use of by me in speaking of the origin of Tubercle, which, I observed at the time, required further explanation. I said that Tubercle was a heteroplastic growth as distinguished from a hyperplastic one. What was my meaning? I must introduce a little physiology and take some backward steps before this can be explained; but the larger and more comprehensive view of pathology then attainable quite justifies the digression, and is indispensable to a thorough discussion of the nature and affinities of Tubercle. Homogenesis and heterogenesis are words which might perhaps be substituted with advantage for hyperplasia and heteroplasia; since the fundamental idea which it is desired to convey by them is like and different generation.

"The history of development," says von Baer, "is the history of gradually increasing differentiation from that which is primarily homogeneous." *Omne vivum ex ovo*, and the yolk is in its early state homogeneous. The cellular pathologists go one step further back, saying the ovum is a cell, and the amorphous blastema neither more nor less than a cell content, in which a peculiar change or segmen-

tation of the yolk precedes a highly heterologous, heteroplastic, or heterogenetic formation of brood cells. This is not cell *de novo*, but the development of cells in the ovum which is itself their mother.

Certainly the strongest position occupied by those who argue for the origin of cells *de novo* in an amorphous blastema, is this yolk argument, which, as we have shown, is at all events equally favourable for their opponents.

Next comes the fact that coagulated fibrine is in parts found substituted by organized tissue; what, however, is taken too little note of is the fact that this tissuification or organization never starts from the centre of the coagulated mass outwards towards the walls, but conversely from the walls or sides, from tissues which become looser and softer up to central parts which are fluid; from walls too in which histological elements are found attached and *in situ* that are exactly similar to those floating free in the central fluid.

Finally, there is the observation offered by Helbert and quoted by Vogel. Helbert placed the clear serum which he had obtained from a blistered skin under the microscope, and convinced himself that there were no corpuscles in it: five or six hours later he examined it again, and now found exactly such cells as are seen upon suppurating surfaces.

The almost insuperable obstacles to be overcome in making such an experiment, the difficulty of maintaining a drop of fluid small enough for one to be

certain that no portion has escaped scrutiny, and yet large enough to be preserved for five hours at a temperature corresponding to that of living tissues and little altered in specific gravity, compels me to set this observation aside as not trustworthy; those too which were made by Kühne in Berlin in 1864, and had a similar object in view, offered contradictory results.

The circumstance that albumen and oil shaken together form an emulsion in which pseudo-cell forms are visible, has been stretched to its uttermost limits as an argument from analogy by Ascherson and Bennett. Their reasoning was this: if by such simple mechanico-chemical means forms so similar to true cells can be produced, why may not real cells with potential endowments be formed in the living body out of like amorphous fluids or blastemata?

Von Panum thus made capital cells out of albumen and chloroform. But the argument so far as its logic is concerned really scarcely deserves attention; it is exactly as if Bazzoni, of doll celebrity, had propounded a wax, paint, and sawdust theory in lieu of the accepted doctrines of embryology, upon the grounds that out of such materials he had been able to manufacture very decent imitations of babies.

For myself, I must confess that I hold thus far with Professor Virchow, in that I have never seen the spontaneous development of nuclei in a free blastema.

Simon, in his Lectures (p. 106), offers cytoblasts as a suggestion in place of development *de novo:* these he supposes to be invisible organic units separate from the first moment of their existence, that is directly after the liberation of the blastema from the blood-vessels. He believes that they neither grow by aggregation, as do inorganic atoms, nor cohere together, but develope from invisible into visible forms; that minute oil-globules next enter into their formation; and that round these centres proteinous growth (the formation of a cell membrane, I suppose) takes place. All this is of course possible, a rational conception enough, but lacking the one thing needful—proof.

Cytogenēsis or cell development appears to me to be conducted upon two principal plans, and I attribute the highest physiological importance to the exact comprehension of them.

The one is the fissiparous or homologous mode, the other the endogenous and for the most part heterologous.

In cells which multiply upon the homologous or divisional plan, the parent cell wall, endoplast and periplast together, enters into the composition of the walls of the several child cells. The separation of these from each other is for us of secondary moment. It is effected in two different ways: 1st, by fission or dipping in of the parent wall about the divided nuclei, and by union or fusion of opposite sides; and 2ndly, by gemmation or budding out

of the cell wall, and by pinching off of the outgrowth.

The point of importance is this, that although increase in size to almost any extent can take place in this way, no higher, no different development is thus attainable; the ultimate products are always facsimiles of their parents. Hyperplasia, or increase in quantity of like forms, only is thus obtained. Examples will present themselves at once to the minds of all who hear me. Cartilage, bone, connective tissue, cells, all increase thus.

But in those cells that multiply upon the heterologous or endogenous plan, the external wall, the periplast of the parent cell, forms no part of the external wall of the brood cells; here too the new or child cells are probably separated from each other in two different ways—1st, by the dipping in of the endoplast about the contents of the parent cell and the segmentation of these thereby; and 2ndly, by the development of new endoplasts and periplasts about the divided nuclei.

Now, by this last method of development the greatest possible differentiation can result, the brood cells possessing different aims and attributes from those with which their parents were endowed: it is thus that the ovum segments produce that most potential blastodermic membrane, thus that the seminal filaments are produced; thus that cartilage and bone, muscle and nerve, develope out of connective tissue; thus that the red blood cell comes out of the white,

that gland cells separate their secretions, and that lymphoid cancer and Tubercle cells are all derived from the connective tissue elements. Yet a few words more. Unicellular organisms, says Huxley, experience only external change of shape, all other plants and animals experience a morphological change of endoplast and periplast. The endoplast grows and divides, but undergoes neither chemical nor structural metamorphosis; the periplast however experiences both chemical and structural change. The chemical changes, which are of two kinds, organic and inorganic, aim at the maintenance of nutrition and at solidification, while the structural changes, vacuolation and fibrillation, are rather directed to the cohesion of parts elementally distinct.

Now, the periplast of Huxley is the intercellular substance of Virchow, and just as growth or development takes place only by means of cells, so the metamorphosis or differentiation into tissue is effected chiefly by the product, excretion, or appurtenance of each particular cell, its periplast or external layer.

This is that which is plastic or shapable—the rest-point, the object aimed at, the form which confers those attributes on a tissue, through which we are enabled to speak positively as to its character, saying this has the features of elastic fibre, that of bone, another of cartilage, of muscle, or of nerve.

I do not for one moment pretend that the cells proper with their endoplasts undergo no structural

change: they are metamorphosed in the direction of the tissue which they compose; they are the method of its nutrition, the means of its growth; and they are capable of functional activity as cells long after they have been considerably modified in shape, whether by flattening or lengthening out in one or more directions. But it is the intercellular substance, the periplast, which by its capacity for further development wields them in its larger grasp and relatively higher bent—this it is which, at first always soft, gelatinous, and homogeneous, becomes granular, streaked, or fibriform, cleaved or split; these are those parts which, although originally separate, fuse together at their edges and confer unity, coherence, and permanency on all formed tissues.

I can now proceed to the affinities of Tubercle with some hope of being intelligible. I have already shown this to be a heterologous growth proceeding by endogenous development out of the connective tissue cells. The young brood do not follow the steps of their parents, or pursue similar structural changes, or become like them; they neither vacuolate in their endoplasts nor fibrillate in their periplasts; they are foci of dissent, which multiply in their turn by continuous fissure, but never return to the perfection of connective tissue elements again; they appropriate nutriment to themselves, grow and multiply in their turn by division; but they are degenerate and short-lived, conceived in haste, and having no power of endurance.

It is in this very matter, the heteroplasticity, as Virchow calls it, of its development, that we perceive a close relationship between Tubercle and Cancer; and, as Mr. Paget has shown in his Pathological Lectures, there exist several other features in which they strongly resemble each other. Both possess a blood dyscrasia peculiarly their own, in both there exists the disposition to get rid of some morbid material by planting it out bad weed-like in isolated groups. Then the body presents sites of proclivity to this "*materies morbi*," and these sites vary with the age of the individual. Both are extra-uterine, never, so far as my own researches extend, congenital diseases; both are strictly hereditary, they also tend to develope in obedience to their hereditary nature at a similar age in the child as in its parent, or at one somewhat earlier. Both infect the tissues in their immediate neighbourhood first of all; both extend through the body secondarily from their respective centres of infection; both induce ulceration, and an ulceration which has little inclination to heal. Both may well be called malignant diseases, I do not mean malignant in the sense of parasitism of the new development—there exists no abnormal growth in the body that is without its normal homologue also in it; malignancy is merely serious heterologism or heterochronism; "elemental parts are abnormal only in the time or place in which they occur or in the method of their grouping[1]."

[1] Virchow's Allgem. Pathol., Bd. i. S. 332.

Still, Cancer as Epithelioma is a graver heterologous error, in its development out of connective tissue, than is Tubercle: the latter has little or no power of maintaining itself, whereas the former is capable of low organization, blood-vessels developing in it as in healthy tissue, and extending freely throughout its substance. The nature of the one is rather to multiply and flourish, of the other to wither and die.

Rokitansky was of opinion that these two dyscrasias exercised a kind of mutual exclusion upon each other, a circumstance which the experience of others has not altogether confirmed. The truth appears to be that the tuberculous diathesis of itself confers no immunity from other disease, and exerts no special excluding influence, but that in proportion as any particular cachexia is strongly marked in one individual, any one error of development widely scattered throughout the body, the proclivity to other modes of structural error is diminished. This at least holds true of Cancer, of Tubercle, of Lympho-sarcoma, of the Enchondromata, of fatty, and of fibrous tumours.

After Cancer the Tubercle growth might be said to present more points of resemblance to the scrofulous tumour than to any other. But of this hereafter; it is advisable for me to defer my description of Scrofula until I have briefly reviewed that group of cognate growths which Professor Virchow has classified together under the head of Lymphatic Tumours.

CHAPTER V.

THE developments to which Professor Virchow has given the name of Lymphatic Tumours possess certain features in common. All are principally composed of lymph cells, these cells being bound together in larger or smaller groups by a reticulum of connective tissue. They are built upon the physiological pattern of ductless glands, and to their proper comprehension some knowledge of the anatomy of these glands is indispensable. The solitary follicles of the intestine and the malpighian bodies of the spleen are examples of the simplest single form; while the compound form is only a repetition of similar lymph cell groups combined together, as these are normally found in the ordinary lymph glands, and in the spleen itself.

There is a dense fibrous capsule which encases the whole, and from this fibrous septa dip in to divide the interior into sepiments or follicles. In the ordinary lymph gland, as will be remembered, these septa form a distinct cortical follicular and a central medullary portion.

This then is the type which we must hold before our eyes. The tumours developed upon this plan possess like elements, and are projected upon a

common scheme, but fall into two clearly distinct sub-classes: the one comprehends the hyperplastic enlargements of parts that pre-existed in a normal state; the other includes new or heteroplastic formations. In both cases the component elements proceed out of connective tissue, and are in themselves identical or highly similar; but the importance attaching to them is obviously much greater in the event of the occurrence of the growth in parts which ought properly to present nothing of their kind.

In the one sub-class, we have the Leukæmic Lymphoma, the Typhous Lymphoma, the Hyperplastic Lymphoma (ex. the simple hypertrophied tonsil), Lympho-sarcoma, Scrofulous Glands; and in the other sub-class, Tubercle of man, and Perlsucht or Parresyge, or Morbus Gallicus, the pearl distemper of ruminating animals.

THE LEUKÆMIC LYMPHOMA.

Under this head are grouped those individually enlarged glands, enlarged by simple increase in quantity of their ordinary elements, which are characteristic of the disease entitled Leukæmia by Professor Virchow, Leucocythæmia by Dr. Bennett. The morbid anatomy of this complaint consists, as is well known, in a general hypertrophy of all the glands in the body, the glandular structures remaining individually perfect and harmoniously proportioned to each other: there is no inclination to death, to caseous degeneration, or to ulceration;

neither do the hypertrophied parts convey ulcerative or indurative tendencies to the tissues about them; the glands remain loose and quite naturally movable in their respective sites, but they are extra large.

The course of pathological changes is for groups of glands—those, namely, which lie in the current of the lymph-stream derived from some one first affected gland—to become involved one after another in similar error. They enlarge, indeed they may be said to aggrandize themselves at the expense of the proper nutrition of the body, and pour the white blood-cells, the products of their elaboration, through the lymph-vessels (these remaining open and perfectly entire) into the general current of the circulation.

The blood becomes deluged with white cells, and mulcted of red ones. Its dyscrasia, or unfitness for the nutrition of the body, and finally for the maintenance of life at all, thus follows upon the local gland mischief, of which it is the secondary consequence, not the primary cause.

The primary gland swelling is not, as in Scrofula, traceable back to any local irritation or skin eruption, but is self-developed.

To us the point of interest is not only the circumstance of the physiology of the tumour showing its kinship to Tubercle, but also because this leukæmic increase in size of minute lymphatic glands,—in places where these are otherwise scarcely visible to the unaided eye, as in the pleura pulmonalis, the

lungs, the pericardium, the kidney, the mucous membrane of the epiglottis, the larynx, the trachea, the bronchi,—these hypertrophied glands, I say, actually come to resemble Tubercle so closely as to have been often mistaken for it: indeed, if the specimen is small, and presented apart from the clinical history of the symptoms manifested during life, the nature of these grey, granular, tiny nodules can only be determined by a minute microscopical examination.

But then the distinction is plainly apparent. The lymph-cells and free nuclei are all sound and persistent in themselves; there is no fat degeneration, no little groups of cells dwindling down to nuclei, and from nuclei in regular progression down again to central granules; and again, the appearances upon mucous membranes are very easily distinguishable from Tubercle, for there is no ulceration present.

THE TYPHOID LYMPHOMA.

The hyperplasia of lymphoid elements, which takes place in typhoid fever in the follicles of the Peyer's patches, in the solitary follicles in the spleen, and in the mesenteric glands, and which becomes the principal local lesion pathognomonic of this particular dyscrasia, is a pathological formation which stands, as Virchow appoints it, about intermediately between the leukæmic and scrofulous lymphoma. It differs from the leukæmic tumour in the proclivity to die manifested by the cell elements

thus unnaturally multiplied, and it approaches the scrofulous gland tumour in this its peculiar instability.

Furthermore, the individual lymph elements present a very apparent deviation from the normal type: they are lymph-cells, but evidently very abnormal ones for their site; they are more cloudy, more finely granular, and much richer in some fat emulsive content than they ought to be. Again, the hyperplasia is to the injury of the surrounding parts. A zone of inflammatory products is infiltrated into the tissues about the swollen gland, and encircles the dead useless lymph heap, itself shortly to be eliminated by a distinct process of ulceration.

The blood error precedes the local lesion, and a corresponding morbid change is impressed upon all the other glands of the body—Virchow's *Parenchymatose Schwellung*, gland cell-like multiplication, which varies in its degree and extent with the gravity of the original blood poisoning. In particular epidemics, too, it is found that the stress of the disease falls somewhat differently upon different organs, although in all the spleen, liver, and kidney present entirely homologous pathological appearances.

The tendency to caseous metamorphosis in the mesenteric glands, which have been the seat of typhous hyperplasia, certainly shows the relationship between this disease and Scrofula upon the one side, while upon the other it must be remembered that the typhomatous, just like the leukæmic gland

hypertrophy, loads the circulating fluid with an undue preponderance of white blood cells.

The typhoid lymphoma does not merely in its composition and course resemble Tubercle; it affects similar parts, it leads to similar lesions—ulcerations in the mucous membrane of the intestine, which, although most often easily distinguishable from those produced by Tubercle by the direction in which they extend, lengthwise rather than encircling, still occasionally offer very difficult questions for morbid anatomists to settle.

In acute tuberculosis the ulceration of the intestine is sometimes so diffusely scattered, so widely extended, and so much on an equality of age or of probable duration, that with no old shrunken scars to guide one by the method of their constriction, and negative or only feeble evidence from other organs, the lesions of the two diseases become by no means easy of differential diagnosis; and the clinical symptoms offered by the two complaints during life are so much alike that the occasional confounding of general tuberculosis with typhoid fever need least of all surprise those who have studied their pathological features with greatest minuteness. Lastly, the kinship of the two diseases obtains confirmation from another source; for the typhoid poisoning is most apt to kindle a dormant tuberculous diathesis into activity.

SIMPLE HYPERPLASTIC LYMPHOMATA.

Under this group are reckoned certain glandular swellings which possess in themselves something of what we term, for want of a better name, a specific nature.

Now the anatomical changes included under this head are extremely various, ranging from mere temporary increase of its lymph cells in a gland up to the gravest possible fibrous hyperplasia and amyloid degeneration. But this need not militate against the utility of the group as a whole for purposes of classification. I have neither time nor intention to do more than just notice them here—a volume might be written upon them. Their variety depends upon the special irritation to which they owe their origin; they comprehend all chronic and acute pathological metamorphoses which are limited to single glands or single groups of glands; as, for instance, the tonsils in tonsillitis and in the scarlatinal lymphoma; the follicles at the root of the tongue in hydrophobia; the hypertrophy, with hyperplastic induration, common to syphilis, which affects, first the glands nearest to the seat of primary infection, and shortly all the lymph glands of the body in greater or less degree, this varying with the malignity of the original virus and the proclivity or impressibility of the individual thus inoculated; and finally, the fibroid indurations, with so-called amyloid degeneration, as found in the spleen, liver,

and kidney, and the superficial lymph glands of the integument, speaking generally, which is found to accompany extensive bone abscess—rachitis, and perhaps some other conditions involving peculiar mal-nutrition of the body.

Now, all these differ from the leukæmic lymphomata by the limitation of the affection to certain parts; and they are distinct from Scrofula, since they are produced by a traceable infecting poison or specific morbid stimulating cause, and because the transition of the glands thus implicated into a caseous state is quite exceptional. They are, further, not simple enlargements in which all the gland elements preserve their correlations, but hyperplasias of some one or other component element; and their development is to the absolute hurt, temporary or permanent, of the functions of the glands thus affected. The kinship of this entire group to Tubercle is anatomically very close: it is also easy to see a relationship in the dyscratic origin of most of these affections, in their tendency to spread by contact, and in the circumstance that so many of them directly predispose individuals to Tuberculosis. They are of the same family, and can further be said to convey some taint of their own cachexia, both to the form and course of the Tubercle growth whose development they favour.

I am by no means alone in my belief that there exists a clinically distinguishable form of Tuberculosis induced in syphilitic and rachitic persons,

which is characterized by peculiar features of rapidity in its early, and chronicity in its later stages; indeed a foundation wash is thus as it were laid on, which lends its predominating tone or colour to the details of the disease as subsequently worked out.

Specific constitutional disturbances, such for example as the influence of vaccination, must modify the entire habit of body; and there is nothing that need surprise us in the extension of this modifying influence to the course and actual anatomy of a new growth, as in tubercular disease.

THE LYMPHO-SARCOMATA.

These are hyperplastic glands, Sarcoma Lymphaticum, the old scrofulous sarcomatous glands. Their characteristic, says Virchow[1], is progressive growth, and occasionally very rapid growth, with persistence of the component elements that thus enter into and enlarge the gland; they infect neighbouring structures by direct continuity, and approximate one step closer to Cancer by their tendency to form metastases in other organs, such as the spleen, the liver, and the lungs, following the order of greatest proclivity in the parts.

The lympho-sarcomata are grey or ash-coloured, and form roundish or very irregularly shaped masses; they attain very much larger size than scrofulous

[1] L. ante cit. p. 729.

glands do, and show little proclivity to ulceration, and still less to caseous degeneration, being scarcely ever metamorphosed into cheesy or cretaceous masses.

They present themselves in two different forms, the soft and the hard. The *soft form* is like the leukæmic tumour, only it does not induce leukæmia; it approximates to medullary Cancer in its alveolar build and in the cell contents which fill its alveoli, and has hence obtained the name of Sarcoma Carcinomatodes. The cells, under the microscope, are small and round with large nuclei, and occasionally some very large ones are seen containing several nuclei in their interiors. The *hard form* consists principally of hyperplastic connective tissue: the normally fine reticulum becomes thick and dense, until the whole gland arrives at a scirrhous hardness.

In the external gland system the sites of predilection are the cervical and axillary glands, and internally the mediastinal bronchial gland system and the thymus: the bronchial gland lympho-sarcoma generally extends into the lung tissue, and that which developes itself in a persistent thymus gland very often implicates the pericardium. A case in which the lumbar and peritoneal glands were involved in this form of growth, and the aorta pressed upon, is narrated by Dr. Ogle in the Transactions of the Pathological Society, vol. xi. p. 251.

SCROFULA.

The old notion of a Virus Scrofulosum has of late

years fallen into disrepute, perhaps for good reasons: it implied that a particular kind of poison was always present in the circulating fluid of scrofulous subjects, a thing which, until this had been isolated, was capable of neither proof nor disproof[2]. Still this idea is retained in a phrase constantly made use of, "blood poisonings;" and if we allow of acute blood poisonings it might be argued, what right have we to reject a chronic attaintment such as is implied in this hypothesis?

The word "poison" might be, and indeed has been, objected to, for it admits of very different interpretations: but by it we take account of any agent that vitiates nutritive interchanges, or arrests or impairs the functions of organs essential to life; and in it we recognize something that can exist latently in living creatures, that can excite regular trains of constant symptoms, that is capable of accumulating in the system, and of being excreted from it. Surely to such an understanding of a poison it might be conceded that the symptoms of Scrofula are not incompatible with those manifested by the body in reaction against some particular virus; and certainly, if the scrofulous poison could be demonstrated in a bottle like vaccine lymph, and be shown capable of inciting special series of lesions that followed its hypodermic introduction with like regularity, we should all acknowledge its title at once.

But since the idea thus implied is at present

[2] Compare Ancell on Tuberculosis, p. 566.

speculative and hypothetical, the term, too, of virus is best abandoned, although the existence of a scrofulous diathesis prior to any manifestation of scrofula must be accepted.

By the expression "scrofulous diathesis" we convey, in a concise and convenient manner, our recognition of a special habit and type of body, such as I believe is characterized by structural peculiarities of form and build.

The scrofulous build of body is an external and visible sign testifying to an inward vulnerability of that body to certain trains of local lesions; and similarly the tuberculous cast of frame indicates a proclivity in that body to develope Tubercle.

Those who have reckoned Tubercle and Scrofula together as merely different expressions in disease of one and the same habit of body, found themselves compelled to adopt two types of scrofulous persons, the very antitheses of each other. The two ideal pictures, however, were indeed so unlike, I may say so directly opposed to each other, that when approximate resemblances became admitted, every form and complexion common to Europeans was included, and the whole Indo-Germanic race might be entitled scrofulous.

Canstatt thus presents us two types of scrofulous persons, the torpid and the fiery. Both are powerful drawings, good observations, and true to life; but I take it they are strictly correct, not as of two kinds of scrofula, but as descriptions of the scrofu-

lous and tuberculous builds of body, which are as distinct as they possibly can be.

Scrofulous persons are large-framed, their bones are heavy, their heads large, their wrists and ankles thick, and their jaw-bones broad. They are big-bellied and coarse-featured. The lobes of their ears are apt to be tumid, their noses and lips often look swollen, their skins are soft and velvety, but their complexions are muddy, their muscles are large, but feel flabby and hang flaccid. Such persons are torpid in mind and body, slow to think and slow to move, being physically incapable of exertions easily endured by those who are of smaller build and look much feebler than themselves.

Contrast with this the tuberculous type—Canstatt's fiery form of scrofula. These persons are tall and thin; their weight is disproportioned to their height, their heads are small, their bones light and long; the ends of their long bones are taper, their muscles are slim and soft; their teeth are thin, long, transparent, and highly enamelled. Their hair is fine and silky, their skins are very fair, and the veins very visible. Their faces are generally oval, their features are delicately chiselled, their lips and cheeks are ruddy, their sclerotics clear blue and pearly, such as bestow a beautiful liquid appearance upon the eye. They are quick and premature in every thing from earliest infancy; they cut their teeth early, and walk before other children get upon their legs. They are precocious as infants;

nervous, irritable, and impatient in childhood; and if they attain to adult years, are distinguished as people of genius or talent, rather than as possessed of particular vigour of mind or profound powers of judgment.

The scrofulous diathesis may be recognized at the outstart as a preternatural vulnerability to certain forms of chronic inflammation, the tuberculous as a special proclivity to one particular form of disease. The first evidence of established Tubercle is the presence of a primary Tubercle new growth; the first evidence of established Scrofula is some glandular enlargement, the scrofulous tumour, which has followed upon some slight irritation of the skin, or some local ulceration.

Now, the anatomy of the primary eczema, or otorrhœa, or ophthalmia, of the scrofulous subject differs in no respect from similar affections as these occur to the non-scrofulous. Sir Wm. Lawrence, discussing Scrofula in his Lectures, acknowledges this in these terms: "There is," he says, "an insensible transition from common to scrofulous disease, and hence the diagnosis is often somewhat uncertain; the ordinary processes are merely modified, the inflammation, instead of being acute, is chronic and leads to glandular swellings and suppuration by a slow smouldering process."

Three things, however, render both the primary lesion and the secondary gland swelling of Scrofula peculiar: first, the facility with which the mischief

is aroused; secondly, the size and extent which it attains; thirdly, the chronicity or sub-inflammatory character of the whole after-process.

Abernethy, and after him Lloyd, attributed the scrofulous diathesis to some original error in the chylopoietic organs. Now, dyspepsia is doubtless as characteristic a feature of this habit of body as are those attributes of form or build which I have already specified. The scrofulous diathesis, like that of Tubercle, Syphilis, and Cancer, is the disposition of body which favours the development of this disease; it may descend as an heir-loom by inheritance, or may be acquired as the consequence of one or more unfavourable conditions of life. This predisposition may be strongly or feebly marked, but its degreee is expressed by such form or build attributes of body as have been already noticed, still it is for many reasons advisable that we should regard it only as a more or less combustible train built into the very fabric of a man's frame, to whose ignition or development some spark or exciting cause is essential. Now, the predisposition may be very great; but yet, if every exciting cause be avoided, the actual symptoms of the disease, the established cachexia, need not, or may only very feebly, manifest itself; and on the other hand, although the predisposition be infinitesimally small, the subject may be exposed to exciting causes numerous and prolonged enough to evoke even this into active being.

The age at which symptoms of scrofula may show

themselves extends certainly over the largest half of a man's life: from the time of weaning up to that of the declension of the generative functions is the wide range allowed by many authors; but we must remember that a great deal of hereditary syphilis and tuberculosis is comprehended in the statistical tables that have been compiled.

Niemeyer[3] thus enumerates the principal circumstances which in his opinion favour the production of scrofula:—

1st. Manifest scrofulous disease, in one or both parents, at the time of procreation, or in the mother throughout the period of pregnancy.

2nd. A taint not scrofulous in kind in the health of one or both parents, such as their being afflicted with Cancer, Syphilis, Tubercle, or Marshmiasm.

3rd. Too close intermarriages.

It is impossible for me, out of the scanty data I possess, to apportion their exact value to his observations. All my own experience inclines me to look upon Scrofula as a distinct disease, with regularly recurring trains of local lesions of its own, and such as manifest themselves only in consequence of an inherited or acquired scrofulous taint: in this particular it is in no way different from Tubercle, Cancer, Syphilis, or Leprosy. Certain particular conditions produce all these complaints. What these conditions are, we at present only partially know, and

[3] Pathol. u. Ther., Bd. ii. S. 743.

certain structural changes which we are studying more exactly, and certain clinical symptoms with which we are slowly gaining better acquaintance, indicate their existence.

Lastly, two or more of these conditions may perhaps occur together in the same individual, and thus render our recognition of them difficult, although altering in no degree their essentially separate natures. Unhealthy parents do not invariably beget unhealthy children; still their tendency to do so, and further to reproduce in their offspring the same diseases as they themselves were afflicted with, is a law as indisputable as that great one of like producing like, which is illustrated by the inheritance of feature, form, and character, of handwriting, of modes of thought, of methods of combined muscular movements, such as bestow similar expressions of countenance on families and races.

Whether or not Scrofula can be acquired when it has no hereditary taint or structural debility to fasten upon, the possibility of its absolute acquisition is not so easy a question to answer. But for our purpose it is enough when we know that unfavourable conditions of life, bad or insufficient food, foul air and overcrowding, if they do not actually engender Scrofula, can kindle its tinder and fan its smouldering flame; and again any fever or accidental hurt which injures the health can develope the same latent constitutional vulnerability. If asked, what then is the reason of this preternatural vulnerability in Scrofula, we can really

only answer, something imperfect in the body. The Humoralists localize this imperfection in the fluids, the Neuropathologists in the nervous system. The Cellular Pathologist discards neither the humoral nor neural view of the question—he says, in my view the blood also is a tissue composed of cells and intercellular substance, only the intercellular substance is fluid; doubtless this tissue may suffer changes which render it unfit to fulfil its functions at all times equally well. And so too of the nerves and nerve-centres—these through defective function or structural error may so influence the digestive organs as to render them incapable of forming healthy chyle, or of converting this chyle into blood, or of elaborating this blood into tissue. But the primary error, the first causes which you fall back upon in both your propositions, is not demonstrable, and merely shifts the difficulties, of which it offers no real solution, further backwards and more into the dark—they are plausible hypotheses created like the atomic theory to meet a certain array of facts, but not equally happy in answering the purpose. Now, on my part I offer you only what you can see for yourselves—a distinct abnormality—something wrong developed or out of place in the part wherein it is found; as to the fundamental cause of this error, I can only derive this from some structural weakness in the cell or tissue that first presents it. In Scrofula this particular weakness of contraction appears to reside in the lymphatic apparatus, and to consist in the vulner-

ability of this to impressions or irritations from without. But you may ask, and very fairly ask, what do I mean by vulnerability?

Habit has been called the memory of the body; and there are habits of right, in the perfect adjustment of supply to demand implied by perfect nutrition, for which mere long continued performance becomes, as it were, a guarantee; since that which has been performed well for a long time, is all the more likely to continue to be well performed.

But while the body is building, growing—a process (and this is a most important point) never carried on after fœtal life with equal activity every where—there is a disturbing agent ever at work; one or other part is not only being maintained at the expense of the nutritive fluid—it is also being further developed and perfected. It is then, I think, easy to see that just this weight in excess of perfect counterpoise becomes an item of possible misapplication; it is the extra strain upon the memorial instincts of the body which cannot at all times and under all conditions be equally well supported. This explains the vulnerability of infancy and childhood to all disease. Again, organs which are at one time in active use, and at another only passively nourished, pass through great and rapid variations of state; this renders them vulnerable. Now, the lymphatic system appears the vulnerable part in Scrofula: the lymph glands and their vascular prolongations are so far weakly constructed that they

get thrown out of gear more easily than they ought. This is that structural debility the existence of which I plead for, and which has been long since allowed by so many medical authors under the insufficiently precise expression of a " scrofulous taint."

CHAPTER VI.

Scrofulous disease is rarely established all over the body at the same time; it is usual for it to waste its force, as the phrase runs, on different parts at different times.

There are three great gland groups which, according to Virchow, are for the most part separately and independently affected—the cervical gland system; the thoracic or bronchial gland system; the abdominal or mesenteric gland system.

For purposes of diagnosis it is also advisable that we remember what Sir W. Lawrence so justly observed, that the glands of the groin are rarely [1], and those of the axilla are still less frequently, affected with scrofula.

This limitation of the disease to certain districts is due to the circumscription of the primary irritation within particular tissues, and to the extension of its influence only through particular channels: these tissues are the intercellular or submucous, and the channels are the lymph vessels.

[1] Curiously enough, since writing this chapter, a very distinct example of scrofulous disease in the inguinal glands has fallen under my notice, in the person of a boy who has quite recently been staying at the Metropolitan Convalescent Institution at Mitcham.

Thus it is that, in scrofulous subjects, a conjunctivitis, a skin eruption upon the head, an eczema upon the external ear or on the face, leads to a peculiar chronic intumescence of the cervical glands; a catarrhal bronchitis to a lobular pneumonia attended with similar or homologous enlargement of the bronchial glands; while an intestinal irritation quickly implicates their mesenteric glands.

It may be said that ordinary inflammations also lead to glandular enlargements. True; but upon the subsidence of the primary inflammation the gland swelling abates; in scrofula, contrariwise, the glandular tumour becomes the principal focus and independent seat of action.

"The gland change consists," says Virchow[2], "essentially in an increase of the cellular elements, and especially of the lymph cells: these, which proceed from division of pre-existing normal cells, are ill constructed from the first, and incompletely organized, not passing into free nuclei only (free nuclei are normal constituents of lymph glands), but forming large remarkable looking cells that fulfil the ideal cell shape much better than those usually found in healthy glands. This is the scrofulous cell *par excellence*, which, like the leukæmic or lymphoma cell, is originally a lymph element: it is a tough, transparent, finely granular, round body, containing one or several moderately large nuclei, and is generally

[2] Op. ante cit. p. 591.

about the size or a little larger than an ordinary lymph cell."

Their peculiarity is their perishability, if I may coin such a word—their inclination to degenerate through fat metamorphosis.

An increase in quantity of such like cell elements produces hypertrophy of the gland and bestows a moist, spongy feel upon it; the capsular vessels become much enlarged, and the gland obtains a reddish colour, passing into a dirty, dead-looking, or opaque whiteness wherever the fat change is furthest advanced.

From this state of things a recovery is perfectly possible, the abnormal elements being completely resolved.

But besides the cell increase, a hyperplasia of the connective tissue about the follicles or sepiments of the cortical portion of the gland is apt to ensue, which outsteps the cellular hyperplasia and leads to intense induration, "adenosis," of the gland [3]; the supply of blood to the capsule is then less liberal, and the second stage sets in, the whole gland becoming thick, tough, anæmic-looking, and dry, from which state it quickly transforms into a yellow, opaque, defunct, cheesy mass.

Three different courses are now open to this case-fied gland—1st, Partial re-absorption. If the blood-

[3] This acorn or chestnut-like appearance on section, recognized by Professor Virchow, is admirable as a description of this state of gland.

vessels remain entire, the greater portion of fluid belonging to the gland is slowly taken up, the cells shrink, the mass dwindles down, fatty compounds, cholestearine, &c., crystallize out in it, lime salts are deposited, and the whole becomes invested with a firm connective tissue sheath.

2nd, Colliquation. The cheesy mass may soften; a change likened by Virchow to a chemical dissolution such as a thrombus, or otherwise insulated dead organic substance, can undergo any where in the body.

3rd, Inflammatory softening. The defunct parts may excite ordinary inflammation about themselves, and pus be formed; the pus-cells proceeding out of the connective tissue by heterologous development, and being evacuated, mix with the other dead elements of the gland.

Such is the pathology of the scrofulous gland tumour which I have described in the way of abstract from Professor Virchow's work on tumours, but sufficiently in detail for purposes of comparison.

Its development is like the typhoma; its component elements somewhat resemble Tubercle, but they are not grouped after the same manner; and again it is a hyperplastic, as distinguished from a heteroplastic lymphoma, an hypertrophy of a normal structure, not a new growth altogether.

I have already mentioned the three great tracts of Scrofula. Now, so long as the glands of the neck, which gave the disease its name in the first instance,

were thus affected, nobody disputed the fact that they had scrofulous disease before them—chronic inflammation, glandular swelling, caseous or cretaceous metamorphosis of the gland, formation of abscess, healing by a slow process, with a tendency to break out again, and with a disposition in the disease to creep insidiously along, and to recur time after time—all this was accepted as Scrofula.

But when an exactly similar process presented itself in the abdominal or thoracic tract, the variation of the locality, although marked by no essential difference whatever in the series of changes or motions, set men thinking that they had a different form of disease before them. The mesenteric gland changes that had followed directly upon a scrofulous folliculo-enteritis were attributed to Tubercle; while in the thoracic tract the implication of the lungs, a gland tissue of peculiar aim and therefore of very special structure in scrofulous chronic inflammation, gave Louis first, then Laennec, and after him the French schools, their primary conceptions of tubercular disease, and, as Virchow and others maintain, a false idea of the constitution and essential nature of Tubercle.

If, as in the neck, the scrofulous process had expended itself less upon its primary seat, and more upon the bronchial glands (which although large do not become proportionately enlarged), its pathology probably would have been more correctly understood. The peculiar, round, millet-seed-like cast of the ulti-

mate alveoli, and finer bronchi, formed of inspissated catarrhal mucus, which shot out like an adventitious product upon gentle pressure, gave colour to the deception, by its likeness to actual Tubercle of the pleura and serous surfaces; and the not infrequent occurrence of the two diseases together in the same person (for they in no way exclude each other) led to this mucus cast of an air sac; for I can call it nothing else, being thought to be the *fons et origo malorum*, the typical and primary form of Tubercle.

Hither came all the students to observe the process of its formation and learn its real nature.

It was an adventitious product—it was produced by inflammation; true enough, it was the result of a local inflammatory process. It was deposited from the blood, and coagulated in the part where it was found; it softened into a cheesy mass, which hollowed from its centre. As Carswell showed, it was the almost entire plugging of small bronchi which gave rise to this idea. Vomicæ were produced by the colliquating influence of the softened tubercles, by the death, from pressure and cutting off of nutritive supplies, of the interposed parts, and extended by the inflammation of surrounding tissues thus aroused. All this was sound observation and perfectly corroborated. The disease affected the apices of the lungs, by preference, and if Tubercle (for which read caseous mass) was forthcoming any where in the body, this part of the lungs was almost certain to afford additional assurance of it. Indeed, to such

importance as a means of diagnosis was this infarction of the apices elevated, that when signs of inflammation were found in the lungs, the situation of the lesion alone was considered quite enough to indicate its nature and origin.

But, so far as I know, little or no explanation, certainly no very satisfactory one, has been offered to account for the affinity of Tubercle, whether real or fictitious, for the apices of the lungs. The vulnerability of serous membranes for Tubercle was accepted as simple fact; the vulnerability of the brain in infancy for Tubercle might be interpreted by the great vegetative activity of this organ at this period of life[1]; but I am not aware that any greater nutritive activity could be allowed to the apices of the lungs than to their bases. Why chronic disease should prefer the apex and acute disease the base—for this is what it comes to, when we reject the Tubercle deposit as a cause and plead Scrofula—remained a recognized mystery: the lung was composed of the same structures throughout—there was no preponderance of one tissue in one part and of another in another. The tuberculous affections of other glands or other solid organs offered no analogy to assist the solution of this anomaly.

But there is an explanation of the difficulty to be found, I think, in the shape and anatomy of the lungs, and in the form of the thorax.

[1] West, Lectures on Diseases of Infancy and Childhood. London, 1859, p. 28.

At the apex, the quantity of lung tissue by cubic measurement, in comparison with the wall area by which it is surrounded, is relatively smaller than obtains at any other part of the thoracic cavity. The condition is that of a small amount of contained substance offering a comparatively large amount of surface.

Pleural adhesions must therefore possess a much firmer hold or grip upon the apex of the lung than elsewhere. Here they tie the parts down as with a tight fitting cap, whereas from the thoracic walls they set a slight limitation only upon its free movements. They must further interfere with the passive act of expiration effected by the innate resiliency of the elastic fibre frame-work of the air cells, much more completely here than in other parts of the lungs; and this for two reasons—1st, because, as I have said, the adhesions must restrict the inspiratory expansion of the apex of the lung; and 2ndly, because when they are old, obsolete, and contracted, bands rather than strings, the lung substance, through which the final stretching has to be distributed, is small in quantity in comparison with what is presented in the thicker portions; and therefore in each individual air cell the component parts must be subjected to greater strain.

Lastly—for it may be urged that deposits are sometimes found in the lungs' apices when no signs of old or recent pleurisy are forthcoming to explain their presence; this is very rare, but still must be accounted for.

How then? I am told by experienced physiologists that there is a normal difference in the size of the air cells in different parts of the lungs, and that the sacs themselves are individually larger and more embryonal looking towards the apex. If this be so, the alveoli must afford more room for the lodgment of catarrhal mucus, and offer individually less counter-elasticity to expel this. Hence the millet-seed-like infarctions will be large and attract attention, and upon section of the tissue they will slip easily out of their moulds, being comparable in every respect to renal tubular casts; but real Tubercle of the lungs lies interpolated in the interstitial peribronchial tissue, and is not thus easily eliminated for separate examination. Finally, the casts themselves, as van der Kolk showed, present a converse arrangement of elements to what is observed in real Tubercle—the largest cells lie in the centres of these quasi bullets, and the smallest at their circumferences.

But apart from the pleurisy hypothesis, the inspiratory dilatation of the cavity of the thorax must, from the anatomy of the normal skeleton, be less perfect at the apex than at the base; for the range of movement allowed to the topmost three ribs is naturally somewhat limited: and again of forced expiration it must be conceded, that the ascent of the abdominal viscera cannot but exert actual pressure upon the lower lobes of the lungs, and so help to squeeze the air out of them.

The corollary to all this is that, in rachitic or otherwise deformed chests, the parts of the lungs whose movements are most limited will be found most prone to be infarcted with thick catarrhal mucus; and that, in spasmodic expiratory efforts or cough, materials blocking up the air cells will be expelled much more readily from the bases than from the apices of the lungs. What I therefore premise of chronic catarrh of the bronchi is, not that the pathological process is peculiar, or that the original lesion falls upon the upper parts and avoids the lower, but that it is generally diffused throughout, and that the external conditions favour a speedier and more complete resolution of the disease in one than in the other place.

I would carefully guard myself from being supposed to urge, that there is no such thing as tuberculous disease of the lung, or to have pretended that all chronic lung affections leading to the formation of vomicæ must be scrofulous, or to have said that Tubercle avoids the apices.

I only plead for a patient reconsideration of the opinion, that pulmonary consumption or phthisis is always produced by tubercles; that the implication of the apices of the lungs is trustworthy proof of the tuberculous origin of an abscess. I believe in scrofulous bronchitis, and scrofulous pneumonia; that is, I believe in chronic forms of both these complaints, such as are easily excited in particular constitutions, and which damage the lung structures very in-

sidiously and are pre-eminently difficult to heal—forms of disease which are attended by early enlargement of the bronchial glands, and their speedy metamorphosis into caseous or cretaceous masses. Lastly, I believe that, although Tubercle affects the lung in the first instance indiscriminately throughout, yet that it is most prone to develope itself in any old puckered scar tissue, if such be present any where.

In scrofulous (grey) hepatisation of the lung, the catarrhal process extends into the ultimate air-cells, the appearances finally attained being scarcely distinguishable from those which attend acute pneumonia; catarrhal mucus-cells and fibroplastic ovoid nuclei are mixed together and block up the alveoli; the intervesicular spaces are trebled in thickness, and come to present a dense and more or less distinctly fibrilated connective tissue.

The difference between the acute and chronic pneumonia is seen in the after stages, the softening and degeneration of the products of the inflammation: this softening in the more chronic disease so affects the framework of the air cells, that the elastic tissue loses its extrusive power, and cannot expel the accumulated secretion, the secretion itself being thick and difficult of displacement.

The consideration of scrofulous disease, whether in the cervical, thoracic, or abdominal tract, from a clinical point of view, offers features which contrast almost as strongly with Tuberculosis as the de-

scriptions already given of the two builds of body. Scrofula is pre-eminently a chronic complaint: the extent of the lesions or the importance of their site can, and of course does, aggravate the symptoms of the disease; but the system at large does not sympathize very greatly with the local affections: fever, or at all events high fever, becomes therefore the exception rather than the rule; further, it is always secondary, never primary fever; subsequent, not precedent to the lesions manifested. The disease is like the hydra, perpetually budding out fresh heads, recurring again and again times out of number; its duration is contemporaneous with the life of the individual, with the exception of a possible interval of quiescence at the period of the establishment of puberty.

To resume, then. The scrofulous tumour differs from Tubercle in its component elements, and in their mode of arrangement. The two diatheses are markedly distinct from each other, the two cachexias are as different as they can be: the one is a disease of long duration attended by only slight sympathetic fever, the other an acute disease febrile from the first, the acuteness of whose course is accurately commensurate with the amount of fever that attends it, this fever preceding the formation of the Tubercles, and lasting throughout the tuberculizing process.

In the scrofulous cachexia the lymphatic system is excessively developed and eminently irritable; the habit of body is sluggish; the reaction against local

lesions appears characterized by no resentment; there seems to be no strength of purpose, no conviction of the right course of their development, or of their proper maintenance inculcated upon the lymph elements; a wrong impression is feebly resisted; the return to a normal equilibrium is slow; the errors that occur are set right with difficulty and after a long lapse of time; the "*vis medicatrix naturæ*" is lacking; the recuperative powers are altogether defective.

But in the tuberculous cachexia the blood appears the seat of graver error; it may be, nay, often is, rich in red blood-cells; the white corpuscles are not formed in preponderating disproportion; the patient looks florid and sanguineous, although the real quality of his blood is exceedingly poor, and its unfitness for the general purposes of nutrition is much greater than could *a priori* have been imagined.

Evidence of this impaired condition of the blood is seen in the mal-nutrition of the body, its leanness, the lightness of the bones, the feeble development of the muscles. Both the voluntary and involuntary muscles show actual deficiency in contractile power; the heart's action is feeble; the intestinal coils are over-readily distended by flatus; the iris is sluggish in its movements (a dilated pupil may mean anæmia only, but sluggish response to the influence of light indicates either muscular incapacity or nerve insensibility); further, as Rokitansky has noticed, in acute Tuberculosis the muscles are dark-coloured

and flabby, and their general parenchyma is infiltrated with serum.

The dry, rough, branlike skin in such persons, the feverish heat of their hands, the tendency which they evince to local congestions and slight hæmorrhages from mucous surfaces, are proofs of this general cachexia, which finally culminates in the building out of heteroplastic new growths, having neither aim nor object, and in parts which ought properly to present nothing of their kind.

Finally,—give it what weight you choose, I attribute very great importance to it,—there is the striking difference in the effects of remedies upon these two diseases. The scrofulous is certainly amenable to ameliorating influences—good air, good food, healthy habits of life, careful treatment, the judicious use of iron and cod liver oil, the removal of sources of irritation, or their allayment as they arise as speedily as possible: all such measures are not without avail—the disease gets better, or may pass into abeyance for a time; the symptoms at least are curable, although the vulnerability, the faulty diathesis, is not got rid of. The extirpation of the enlarged and useless glands, which having ceased to be blood factors have become blood poisoners, where this can be effected without danger, should be done early. The results of this plan of treatment in the hands of Langenbeck and others were certainly encouraging.

But all this is quite opposed to our experience of

tubercular disease: here we may foresee the evil afar off, but just as with cancer our foreknowledge helps us not one whit—being forewarned we are not forearmed against the storm when it arises. To procure a euthanasia is perhaps the only endeavour really attained by the physician.

The difficulty, however, to be met in distinguishing Scrofula from Tubercle, is that scrofulous persons perhaps sometimes become tuberculous: the two diseases are not incompatibles—they exercise no excluding influence upon each other, although themselves specifically distinct. Nay, there are even points of resemblance between them, just as there are between lymph-cells and tubercle-cells, and between cancer and epithelium-cells; but there are also features of sufficient difference to authorize us in drawing a line of demarcation between them. Nothing is easier than to perceive inter-resemblances, nothing more difficult than to make out titles of identity. The man who is brought low by fever, by measles, by chronic disease, by diabetes, by scrofula itself, becomes in some instances the subject of tuberculous growth; in like manner as he who has suffered with successive fibrous tumours, at length developes a medullary cancer. The two diseases may be fairly called allied—they are genera of one large family, the lymphatic family of disease—and yet clinically and pathologically they are distinctly separate. The one is especially an acute, the other a chronic disease; the

one the complaint of a life-time, the other the sudden disturbance of a life, induced often by some great subversive change in the conditions of climate, food, or clothing to which an individual is subjected.

It is contrary to all our definitions, all our ideas of scrofulous disease, that a man should reach eighty years of age and then manifest scrofula for the first time—a circumstance which must be allowed if newly developed Tubercle has been found at this period of life, as has been asserted, and if Tubercle and scrofula be different manifestations of one and the same cachexia.

CHAPTER VII.

I PROPOSE next to give a general review of the clinical history of Tuberculosis, and to discuss the local affinities of Tubercle.

By Tuberculosis I understand that process of disease which is attended by the formation of Tubercles. In the first place must be noticed the proclivities which individuals manifest for developing Tubercle. After what has been already said about the tuberculous diathesis, it will not be necessary for me to say more than that this habit of body is both inherited and acquired. The circumstances favouring its acquisition are much better known and authenticated than in the case of scrofula. For examples of this acquisition of the disease when the hereditary taint was an inappreciable quantity, I must refer to M. Leon Colins, *Etudes Cliniques sur la Tuberculisation aigue.* Several cases are there adduced—and very admirably detailed they are—illustrating the occurrence of acute Tuberculosis in the persons of soldiers who had returned to France in the winter season after living some time in Algeria. They are the converse experiments to those ordinarily offered for illustrating the influence of climate in arresting or curing tubercular complaints. M. Colin says, "The consideration of

these cases leads me to conclude that there are two principal forms of acute Tuberculosis—"l'une primitive, survenant chez un sujet sain, à l'instar d'une pyrexie par exemple; l'autre secondaire, se manifestant chez un sujet préalablement tuberculeux. Cette distinction n'a guère été relevée par les auteurs classiques que pour la meningite granuleuse." Now, if the tuberculous diathesis is plainly expressed, this general implication of so many organs together, or one after the other, of the pleuræ, lungs, spleen, lymphatic glands, intestines, and the membranes of the brain, attracts sufficient attention to make the diagnosis tolerably easy; whereas, if the affection commences with sudden invasion, and there is no history of hereditary proclivity, no tuberculous aspect or build of body, to afford any clue to its interpretation, such a case, which may run its course to death in from eight days to two months, offers one of the most puzzling questions that we have to solve at the bedside. Such cases are frequently mistaken throughout for typhoid fever, which they simulate in no small degree.

The illness begins suddenly with rigors; the temperature of the body rises quickly, and remains at or about 103° (Fahr.). There are sudamina upon the skin, and it may be a few rose spots over the trunk [1]. The spleen is enlarged, the tongue dirtily furred, inclined to dry at night-time, and sometimes

[1] Vide Obs. 6.

to remain dry and fissured by day, but neither sore nor particularly red at the tip or edges; the appetite is often preserved close up to the end of life; menstruation too continues to be regularly performed; the bowels are generally confined, but not tender to pressure, and there is no meteorism; ulceration of the intestines, if such be present, is not confined to the Peyer's patches, but is widely diffused, and comes on as a late symptom, secondary for the most part to the tuberculous implication of the mesenteric glands. The principal symptoms vary of course with the involvement of those different organs upon which the stress of the disease falls—as the kidneys in one of M. Colin's cases (7), and the brain in another (the case of Warpot). But the principal circumstances that assist us in making a differential diagnosis between acute Tuberculosis and typhoid fever, are the nature of the delirium and the degree of prostration of the patient.

In acute Tuberculosis the delirium is rather characteristic: the patient is generally rational and sensible by day, towards night-time is apt to pass into a dreamy muttering state, groaning a good deal, though capable of being roused, and then not complaining of much pain, he rarely becomes violent or attempts to get out of bed; again, although he wastes rapidly, and is often sick and unable to absorb what food he takes, his condition is seldom so adynamic that he is incapable of moving himself in bed; he is conscious of his wants till quite the last, and does

not contract bed-sores. All this contrasts very strongly against the utter helplessness and frequently furious delirium of persons in typhoid fever.

Further, acute Tuberculosis is quite distinct again from what is vulgarly called galloping consumption, in which disease all the usual symptoms of a scrofulous lung affection are presented, only succeeding each other with extraordinary rapidity.

The pathology of acute Tuberculosis is the very general and diffuse development of the tubercle growth in almost every organ of the body. If the peritoneum be affected, this membrane, as I have previously described, becomes covered with little granulations, and is enormously thickened, so as to be like the flesh of a chicken (chair de l'oiseau), as Trousseau was wont to call it.

In the spleen, the tubercles are found occupying the Malpighian corpuscles themselves; the kidneys are generally riddled throughout with them, and so is the liver, both in its substance and upon its peritoneal surface. The lymphatic glands are the only parts in which great hypertrophy is reached; but these often become enormous, and lead to the compression of bronchi and mesenteric arteries, and sometimes to the occlusion of the ductus choledochus.

The lungs, however, are not more affected at the apices than at the base; they are usually gorged with blood and seem swelled; the pleural membrane is mostly covered with grey granulations, and similar

appearances are found scattered throughout the lung substance, especially in the course of and within the outer sheath of the smaller bronchi.

This acute acquired Tuberculosis appears almost as an epidemic disease at certain times, whereby its affinity to typhoid fever is still further indicated: it occurs to delicate persons, and is mostly excited by some sudden change in life, as from a residence in a warm, dry climate to a wet and cold one. In Europe it presents itself usually in the autumnal months of the year, in October and November, and principally attacks young men and women in the first two or three years after the establishment of puberty—indeed, just at that time of life when I should say that real scrofulous disease was most in abeyance.

In particular epidemics the stress of the local lesions should appear to fall upon like organs, at least if we are justified in making any deductions from the scanty evidence which we possess, as in the epidemic of 1863, narrated by M. Colin, upon the membranes of the brain especially, and in that of 1865, upon the lungs.

It must be confessed that we possess no sufficient data to enable us to speak positively as to what conditions of body most principally favour the development of Tubercle.

Atrophic and weakly subjects are certainly, according to our daily experience, more prone to this disease than the robust and strong. But does

weakness and atrophy comprehend the sum of our knowledge of the tuberculous diathesis?—are they not mere manifestations of proclivity to all disease?

The subjects of fatty degeneration of the liver and kidney, and of atheromatous disease of the blood-vessels, have been supposed to be peculiarly prone to Tubercle. Our experience, however, without any trustworthy statistics, allows us neither to affirm nor to deny this.

The predisposing causes of Tuberculosis are chiefly any total change of habits, of diet, or of the mode of life which has been common to any individual, bad air, ill ventilated rooms, sedentary employments, the crowding of numbers of people together, improper or insufficient food, exhausting overwork of mind or body, mal-hygiene in one word. All these, however, are depressing influences, such as facilitate the encroachment of disease generally. They are the collecting together of fuel, the stacking of damp hay: if you leave it alone, it will burst out into spontaneous combustion; if you approach it with a spark of contagion, this will spread through the heap like wildfire.

Wunderlich concluded that typhoid fever, measles, influenza, and diabetes were the diseases the weakening influence of which especially predisposed to the development of Tuberculosis; but under this head he certainly has included some forms of scrofulous disease.

Rilliet and Barthez imagined that vaccination

rendered persons more prone to Tubercle; because in 208 bodies of vaccinated children 66 per cent. manifested Tubercle, while of 95 unvaccinated 31·5 per cent. only were tuberculous. But it is obvious here that the numbers are insufficient to justify the inference drawn from them. Mr. Ancell groups the statistics of scrofula and Tubercle together, and concludes that approximately one third of the cases derive their origin from hereditary transmission.

The whole question of hereditary influence requires more accurate data than we possess for its settlement. Still the impression left upon our minds by the statistics which have been already compiled by Lebert, Rilliet, and Barthez, and with singular care and accuracy by Mr. Ancell, amounts almost to the conviction, that the strongest of all predisposing causes is the inheritance of a tuberculous taint.

The records of life insurance offices, and the registrar sheets of large hospitals, ought to throw more light on this matter.

One difficulty is, that the mortality from this cause, even though exactly determined, would represent a minimum frequency only of the disease; since it is assuming the major premiss to say that all who are thus attacked die; and yet you can obtain no proof unless they die, and no sufficient proof, except all who are certified to have thus died have been examined by persons competent to give an opinion upon the cause of their death.

The strongest argument against an hereditary transmitted taint being the sole or even the chief cause of Tubercle is, that the disease itself shortens life, and therefore limits the multiplication of the human species, and should tend to die out of itself, by a species of natural extinction. The weak element in a race, that which is less capable of transmitting vitality to succeeding generations, must become obliterated altogether after a certain number of generations. And, unless the disease was acquired afresh from time to time, it must grow rarer, from age to age, instead of being, as it is, more frequent, increasing rather than diminishing with the advance of civilization.

Tuberculosis, says Virchow, is essentially a disease of extra-uterine existence (p. 718); and when it is hereditary it is not congenital. After all that Mr. Ancell says of Tuberculosis in the fœtus—and he says a great deal—it must be remembered that he adduces no single example in confirmation of the fact itself (although some, perhaps, in favour of congenital Scrofula).

It still remains for us to discuss the hereditary affinities of Tubercle; and first we may observe that, as with gout and other disorders, it is found sometimes to skip a generation, and reappear in the grandchildren. Statistics incline us to believe, that sons inherit Tubercle more from tuberculous fathers, and daughters from tuberculous mothers; that

the susceptibility or vulnerability to Tubercle is, speaking generally, greatest at the earliest periods of infancy, and that it diminishes with advancing years. Youth and childhood are more vulnerable because the tissues are then softer, and because, as we before said of Scrofula, the habits of right have been inculcated for a short period only.

According to Wunderlich, the tuberculizing process first developes itself with the appearance of the first teeth: it is a very rare event to find Tubercle, he says, before the third month of life. This is the first minimum.

Up to two years of age it affects the lymphatic gland system, much more than the lungs or brain. From two years to fourteen years it makes the first maximum period, allowing a slight relative abatement for the eighth year of life: during this period, if Tubercles be present any where in the body, confirmatory evidence is pretty certainly forthcoming in the lungs.

From the establishment of puberty up to the twentieth year of life occurs the second minimum of death from Tuberculosis; and from the age of twenty to thirty occurs the second maximum. At least half of all the people who die between these last ages present Tubercles in their bodies, and mostly in their lungs.

From the age of thirty there is a gradual decline in the numbers again, the deaths from this cause

attaining their minimum somewhat abruptly between the years of fifty and sixty-five. Still the immunity then appears, statistically, to be rather less than that conferred at the second minimum, the first years after the establishment of puberty.

CHAPTER VIII.

THE discovery of that "little rift within the lute, that by and by will make the music mute," the first cause of Tuberculosis, may be said to be as far off as that of original sin. But we may fairly inquire where this vulnerability of the body to Tubercle is first manifested? Is it the blood which, by its vice, or inaptitude, or deficiency, determines the local disease? or are they the tissues which, by their structural debility, are rendered prone to the tuberculous error?

What is the first sign of the disease, its mark? It is a local fluxion or congestion, a particular kind of inflammation, if we adhere to an old word; a special form of "Reiz," if we borrow a new one from Professor Virchow, which fulfils our requirements in the shape of a term exceedingly well.

The old authors referred every abnormality in the tissues, whether manifested by hypertrophy of pre-existing elements, hyperplastic or heterologous new growths, to inflammation as their direct cause. There were adhesive, suppurative, purulent inflammations, acute and chronic inflammations, active and passive inflammations.

As to the former, the "calor"—"rubor"—"tumor" requirements were readily forthcoming, and the

phlogistic process presented some feeble similitude to justify the use of the term. This justification, however, does not extend to the expanded idea; the term Inflammation is not merely inappropriate, but strictly untrue, of a process into which neither "rubor," "tumor," nor "calor," necessarily enter.

No wonder that this chapter upon inflammation became so large a one: every action of living cells, every metamorphosis of tissue, natural growth and nutrition, natural degeneration and decay, were colligated with equal justice under this comprehensive heading.

There must remain a large number of forms of disturbed nutrition in which the rapidity of the tissue changes, the surrounding blood-flux, the reaction of the neighbouring parts, and the co-suffering or sympathy of the whole body with the local process, will render the application of the term Inflammation appropriate enough. But there are also many growths which, from the first moment that they are recognizable as deviations from normal nutrition, up to the period of their full development, never present any strictly inflammatory phenomena.

It is for them that Virchow proposes the word "Reiz," *irritatio*, to express some cause for this departure from healthy nutrition; the fact being, that the disposition to go wrong, equally with the inclination to go right, is situated in the cell elements or intercellular substances. These are at least higher orders of things than the more succulent nutritive fluid which they determine in some measure towards themselves

by their own attraction for it. The blood does not first push to a part loaded with a burden of good or evil for it; there is a little free will even here. No disturbance of innervation can be the first cause of a new growth; nutrition can go on independently of nerves altogether. The nerves and blood-vessels are co-operative conditions of life, necessities not of, but towards nutrition; and if one must name a part in which a determining power exists, this can only be appointed where some discriminating influence is recognized in health, and where error is first apparent in disease.

Still you ask what is meant by "Reiz;" *irritatio* does not Latinize the word, and I know of no English one which conveys all the sense that Virchow attaches to it. But I think I can give some explanation of its meaning.

The nutritive fluid acts upon the healthy parts to which it is supplied, and is re-acted upon by these in return; so that in normal nutrition there is a balance struck: there being a certain weight or moment for consideration at both ends of the scale, this re-action at the developing parts of the cellular structures (the connective tissue elements, as in the example of Tubercle and Cancer) is the "Reiz" of normal development, and the "Reiz" of abnormal development implies a similar reaction at the part expressed in different or undue degree. The new growth or abnormality may be said to represent not only the amount of difference between the sup-

ply of nutritive fluid and the proper demand for this, but also the x, or unknown quantity of improper demand, the amount and specific character of undue "Reiz," which determines a supply of nutritive material in excess of the equipoise of proper nutrition. Increase of normal reaction implies increased nutritive supply and simple hypertrophy; and different kinds of "Reiz" are convenient first causes to fall back upon for an explanation of different modes of wrong development in cells.

It is quite allowable to suppose that a peculiar stimulation, a tuberculous "Reiz," first takes place in the connective tissue cells, that these are induced in consequence to develope wrongly, to heterologize after a particular manner, and to form a Tubercle. The growth proceeds rapidly, there ensues an increased blood-flux, a local congestion; nutritive materials are thus supplied in extra quantity close round the new growth, although no capillaries actually dip into its structure.

The proclivity to error in the connective tissue cells, their vulnerability, is either the result of an inherited structural weakness, or is the consequence of an acquired debility. But given this beginning, at best only an hypothesis, a convenient theory for a first cause which tallies well with what we know of ordinary nutrition, and holds equally good of Cancer and all other growths,—for the after-history we have an array of observed facts.

The actual exciting cause is a single one, innate

in the cells themselves, a spontaneous tuberculizing error; and for those examples of multiple crops of new growth springing up all over the body at the same time, as in acute Tuberculosis, we need seek no further for any other; but in far the larger number of cases we also observe an extraneous inducing influence at work, some irritation from without, very readily traceable—a mental shock upon the brain—a cold blast upon the air passages—a blow upon the knee-joint, which has evoked an ordinary reaction, or simple inflammation, to which the tuberculizing error has been superadded. But it would be quite wrong if we assumed from this that Tubercle was only produced by inflammation in a debilitated subject. Certainly we should allow due importance to this more frequent origination of it as the sequel of ordinary inflammation: it is this circumstance that makes the physician, with perfect truth and upon the soundest experience, believe that the *momentum excitans* is capable of being avoided, and leads him to enjoin the strictest care in diet, in clothing, and in general hygiene, upon those whom he perceives, by their build or their inheritance, to be especially vulnerable to Tuberculosis.

Again, the proclivity of youth and infancy receives further illustration from the affinity of Tubercle for false membrane and granulation tissue, which is composed of young embryonal connective tissue elements. Now we find that Tubercle is a most

rare occurrence in certain parts of the body, and that certain others present a peculiar affinity for it; and further, that when it originates in particular organs, it extends secondarily through particular tracts.

In thus possessing favourite sites it is like Cancer and Scrofula. Tubercle is very common as a disease in serous membranes, in the testicle, the kidney, the fat tissue of the omentum, and in the medullary tissue of bone; but it is rare in the muscles, in the mammary gland, in the ovaries, in the thyroid gland, in the tonsils, in the salivary glands; very rare in the mucous membrane of the stomach; and, so far as I know, never found in the œsophagus.

It is a question rather with me if it be so rare an error in the connective tissue of the skin as has been asserted: the likeness between the tubercular form of Leprosy, or Spedalsked, and actual Tubercle is so great, that I cannot distinguish any difference between the two diseases; indeed, I believe them to be identical, and that tubercular Leprosy is the peculiar manifestation of this growth in the skin tract. We find that many of those parts which are indisposed for Tubercle are predisposed to some closely allied disease, as the ovary and thyroid gland to colloid or cystic disease; and we further perceive, not only that similar anti-hygienic conditions of life induce these two complaints, and that they have a tendency to occur in the same individuals and at the same time, but that the predisposition towards

them appears conveyed by the same hereditary taint.

The dissemination of Tubercle affords us some insight into the general pathology of this disease. It is quite exceptional for the new growth to develope itself all over the body at once, as we have already described that it sometimes does.

The old idea entertained by Laennec was, that there were primary and secondary eruptions of Tubercle, that the primary nodules were nests of infection, that their infectibility began from the moment of their softening, and that the course of this infection was clearly traceable in certain tracts. He taught that, if the primary affection took place in the intestines or mesenteric glands, the secondary eruption appeared in the lungs [1]. He believed that a specific tubercle substance was transported like a cancer germ from one part of the body to another, and that the means of transport was the blood. Buhl [2] went further than this; he assumed that the caseous products of chronic pneumonia, re-absorbed into the circulation, were converted into tubercle virus, and that it was this that excited the miliary growths: it is Von Dittrich's and Addison's view of the origin of Tubercle over again; namely, that this is produced by a retention in the blood of the products of the retrograde metamorphosis of the tissues.

[1] Laennec, p. 223.

[2] Zeitschrift für Rationale Med., 1857, Bd. viii. S. 64, 68.

The theory, however, is not adequate to all the occasions. In acute Tuberculosis, without any local abscess, or any such source of primary infection, we find new growths of apparently the same age and date springing up in various and distant parts: be it granted that this is very rare, still the one single instance would be enough to prove that Tubercle was not then produced by the re-absorption of retrograde tissue products into the blood. Further, it becomes pre-eminently unlikely that the same growth should be produced by one cause at one time, and by another at another. The hypothesis must meet all the occasions better than this one does, before we are justified in accepting it; and then there remain negative facts to be got over. In the scrofulous gland affection in lumbar abscess, in empyema, we observe that the products of dead nitrogenous tissues are retained in the system for an indefinite length of time, and yet do not always, or necessarily, induce the formation of Tubercle; and further, if more evidence were needed, there are the inoculation experiments. Scrofulous material and tubercle substance must through scratches have been introduced over and over again into the systems of persons who are daily occupied in making post-mortem examinations, but it is not found that the individuals who are thus exposed contract Tuberculosis.

Let it be allowed that it is a very different thing to poison one's finger and to have decomposing ani-

mal matter injected into one's blood: it is certain that by this last method we may excite metastatic abscess (*vide* experiments made by Mr. Savory upon dogs [3], and M. Villemin's experiments upon rabbits [4]), for I cannot accept them as proving the production of Tubercle. Then Erdt, too, injected human scrofulous matter into the veins of horses, and thus excited a disease indistinguishable from glanders [5], and Kortum [6] actually experimented upon children with the secretion of scrofulous ulcers. But it is by no means proved that Tubercle is thus producible at will. What, however, we may fairly allow is, that after typhoid fever, surgical wounds, the post-partum state, measles, and influenza, whether through the circulation in the blood of materials that ought to have been excreted from it, or whether through the conveyance into it of feebly structured white cells, which are incapable of further elaboration, the general nutrition of the tissues is gravely interfered with, so gravely that metastatic abscesses may arise. But then this is all; the abscess does not present the identical features of Tubercle.

It is a matter of common observation that, when the change for improvement has taken place in

[3] Barth's Hospital Reports, vol. i., p. 112.

[4] Comptes Rendus, tome 61, 1865, pp. 1012—1014.

[5] Erdt, Die Rotzdyscrasie u. ihre verwandten Krankheiten. Leipzig, 1863, S. 151.

[6] Kortum, Commentarius de vitio scrofuloso quique inde pendent Morbi Secund., T. i., p. 218, Lemgo, 1789.

typhoid fever, the face and body show signs of emaciation setting in; the urine, (which may be reckoned as some measure of the tissue waste,) which was previously excreted in excess, becomes greatly diminished in quantity [7]. So long as the fever was at its height, this thinning of the face scarcely attracted attention; but now, when the retained water begins to drain off, this loss of solid substance is at once appreciated. The process of repair begins: and how does Nature set this about? By making what materials she has go farther and last longer than they normally should.

Not only the excessive, but the ordinary waste is diminished, and this, although there must be foul, over-used nitrogenous substances retained in the system. In the fourth week one-third to one-half the normal standard of urine excretion only is reached. Perhaps in this fact, the retention in the body of what ought to be excreted from it, lies a predisposing cause of Tubercle, and some explanation of the proclivity to its formation doubtless conferred by typhoid fever. But it is a great jump to conclude that Tubercle springs up spontaneously out of this hot-bed. Here is merely the cucumber frame and the congenial soil. There will be no fruit without seed—no Tubercle growth except by heterologous development out of connective tissue, and this obtains no actual explanation from the circulation of morbid products in the blood.

[7] Parkes on the Urine, pp. 244, 245.

The dissemination of Tubercle, however, from the sites of its primary formation was a good observation, and true; none more so. In this respect Tubercle is like Cancer, like Scrofula, like all the other lymphomata: it is eminently infectious. The prostate infects the vas deferens, the epididymis, the substance of the testicle, the bladder also, and the urinary tract in the other direction. The intestines infect the lacteals, the mesenteric glands, and then the lungs. The solitary conglomerate in the brain substance infects the membranes and destroys life by a general meningitis. The pleural membrane infects the lungs, and thence the liver and the kidney. All this has been admirably described by Virchow [8].

It would be very satisfactory if the law laid down as a great generalization by Mr. Simon, in his valuable Lectures upon Pathology, could have been confirmed, that the organs in which the primary deposits of Tubercle occur were always those that could be called organs of blood-development; but such are not the prostate, the brain, the knee-joint, or the testicle: if we were able to assure ourselves that the error in these parts originated in some lymphatic gland or channel, then this might be accepted as a most important dogma; but this is as yet not proved. Let us refer to Rokitansky's list for the relative frequencies in different parts of the body. First in frequency for *primary deposits* come

[8] L. c., p. 726.

the lungs and lymph glands; 2ndly, the urinary organs, the female sexual mucous membrane; 3rdly, the bones, testicle, prostate, and seminal vesicles. And for secondary deposits, in the order of greatest frequency, come the intestines, larynx, trachea, serous membranes, spleen, liver, and the kidney.

There is a primary Tubercle, and there is a metastasis, or secondary distribution, from this site of infection. Are we taught nothing by this fact? I think that we obtain some therapeutic indications from it of no mean kind. A local irritation, some special "Reiz," excites the first Tubercle into existence; this is accompanied by febrile disturbance and general sympathy throughout the body: surely the object in treatment must be to allay the local disturbance, and so to limit the first infecting source. For scrofulous disease, good food and plenty of it, stimulants and tonics, may perhaps be recommended from the very beginning. But in Tuberculosis the hand of the physician must be guided by some experience of the symptoms of the disease and some knowledge of its pathology—the one simple rule of brandy and food will not suffice here: irritation has to be arrested, and the general irritability lessened; any thing that can excite inflammation must be at once removed—indeed, the local affection, the infecting source, if this be a joint or an external part that can be taken away, is, perhaps, best excised as quickly as possible.

When the primary mischief has abated, and the general fever passed away, attention can be directed to diminishing this special vulnerability of the system. The nutrition may be improved by the careful administration of the simplest and most easily assimilable food. Cod liver oil has been found about the most useful medicine that can be given; tonics must be recommended with great caution; quinine is often only borne in conjunction with a saline like nitrate of potash, and iron should be prescribed with the greatest care: I know of no medicine that is so liable to do harm by determining local congestions and inducing hæmorrhage.

The principle of treatment is to combat the symptoms as these arise, and especially to enjoin an avoidance of any sudden change of climate or of habits of life.

The most beneficial climate is generally that in which the barometer and thermometer experience smallest variation through the twenty-four hours, and throughout every season of the year. Mountainous districts, such as are cold and not too moist, frequently benefit those who are afflicted with scrofulous lung abscess: but temperatures which are low, and variable seasons, do not suit tuberculous subjects.

My text, however, was the nature and affinities of Tubercle; and it is within these limits that I would restrict myself. It has been my object to show, that Tubercle is a new growth, and that its nature and affinities appoint it to a position between Cancer and

the lymphomatous tumours. I would have it remembered that pathological formations are not to be classed in the same category with parasitic disease; that nothing alien to the body is ever grafted, as it were, on to it; that there exist no such things as cells pathognomonic of Cancer or of Tubercle.

New growths are built up after normal physiological types, the structure perhaps most like Tubercle being a lymph gland.

I have followed Professor Virchow in my description of a family of diseases that affect lymph structures, all of which present certain affinities to each other, and which, when grouped together beside Tubercle, afford us a clearer understanding of its nature, and certainly help us to a better comprehension of its real position in the scale of morbid products.

Finally, I have drawn a distinction between Tuberculosis and Scrofulosis, which I believe to be thorough and complete, and clinically of high importance to us, as physicians, when we are forming our prognosis of disease at the bedside, and directing our counter-measures of treatment against it.

But it is also my hope that we have learnt more than this; that we have been looking together through a glass, dimly it may be, but still gaining some insight into the great scheme by which decay, degeneration, death are brought about.

In nature all is orderly; she does not build by laws,

and destroy by their summary abrogation, shattering her edifices by sudden and overwhelming annihilation; she pulls down brick by brick, to follow up our builder's analogy, appoints resting-stages, temporary conduits of supply, temporary channels for the removal of *débris*. We must watch all her steps, study all her operations, observe her master-hand in the conduct of repairs, if we would practise our prentice fingers in the work of reparation.

The laws which order the development of the body are the same as those which govern its errors in disease: one formula must, and will, serve for the solution of both problems, how we live and why we die.

Now, as it is from known laws that we soar to the discovery of new and unknown ones, let us be patient in investigation, slow to theorize, just in judgment upon what is guess-work and what is fact obtained by sound induction, and be true to ourselves in an endeavour to discover truth.

Oersted has well said [2], that "nothing is invariable in nature but her laws; these all effects obey, and stand towards in the same necessary connexion that one axiom in reason does to another."

"There exists," says Powell [1], "a strong natural propensity in the human mind to draw hasty inferences, to generalize rapidly, to deceive ourselves by erecting conclusions upon very unsubstantial and

[1] The Soul in Nature, p. 23.

[2] Unity of Worlds, p. 14.

insufficient data." Let us be very careful in forming inductions, and always ready to correct them when enlarged experience, or further experiment, attests their imperfection. Let us reason from the facts up to the causes, and let us be careful not to accept the mere recognition of changes, that is the re-statement of the case in different words, in lieu of explanation. The attempt to mask ignorance in a cloak of empty words will profit modern physiology no more than it did philosophy of old.

We live in an age of haste, move quickly, waste fast; our population increases, but the mean average individual duration of active life diminishes. This has advantages; it has also its drawbacks, one of which certainly is a tendency to the neglect of accurate and philosophical principles of research. We are in danger of casting away experience as a thing of too slow growth for us, and of laying aside experiment as an expensive touchstone which we can no longer afford to use.

It is impossible to ignore this circumstance, but at all events let us shun the imputation of cant, and avoid that pitfall of metaphysical phrases which has been so fatal to the advance of science in every period of history.

Why formularize the evolutions of matter as "forces"? Does "formative force" explain why or how tissues form? Does "germinal force" help us to understand the development of one cell out of another? Does "vital force" assist us to an inter-

pretation of the actions and motions characteristic of life? Do we gain any clearer conception of the animal creation by speaking of animals (as C. Schmidt does, in his Anatomy of the Invertebrata) as "cells plus soul atoms"?

This mode of dealing with Nature's most difficult problems is not only unwise, it is unfair: if we avoid puzzling ourselves by it, we certainly lead those who are less able or less inclined to reason for themselves, into puzzledom, and leave them there.

The true dynamic is by such language only shifted a little further out of our reach.

There is nothing to be ashamed of in acknowledging our ignorance of first causes.

The elements in living matter may be definitely arranged in suspension of their affinities by a vital force[2]; this force being used in the stead of an explanation to express our recognition of the fact that the ordinary and expected chemical decompositions are suspended, and that certain other peculiar interchanges, such as we are quite unable to imitate in our laboratories, take place.

There is no great harm, although surely no great gain, in our saying that living matter exists in a state of tension. The materials of which the tissues are composed can be said to be held in a vigorous life-grasp in health, to be less vigorously

[2] Dr. Lionel Beale, Medical Gazette, 1867.

grasped in disease, and we might fairly follow out the simile so far as to say that they are dropped out of hand altogether at death. But this is poetry, not philosophy, and such phrases do science real harm; for they open the door to doctrines and theories as speculative as they themselves are vague. It is out of such fancies, in place of facts, that hypotheses of disease are formed, which are blown to the four winds of heaven by the breath of common sense, and which do harm in themselves and bring grave discredit upon our profession.

The propounders of such crude doctrines thus convey to the world the guesses of their leisure hours, rather than the articles of their innermost belief, and usually dress their ideas in language studiedly obscure.

Now, to quote from an old divine[3], "Certain is it, that all that truth which God hath made necessary, He hath also made legible and plain; and if we will open our eyes, we shall see the sun."

Physiology, if it be ever to profit us, should be the λόγος προφόρικος φύσεως, rather than the λόγος ἐνδιάθετος. We have to deal with the simple exposition of the phenomena presented to us by living beings, to learn the order, the interdependence, and the method of their association; and we shall find enough to do, if we labour rightly to understand the facts themselves, without indulging in an attempt

[3] Jeremy Taylor.

to comprehend those first essences or directive agencies which have been placed beyond our ken. This is the *Divina ratio rerum*, and for it let the *Divina voluntas* suffice.

THE END.

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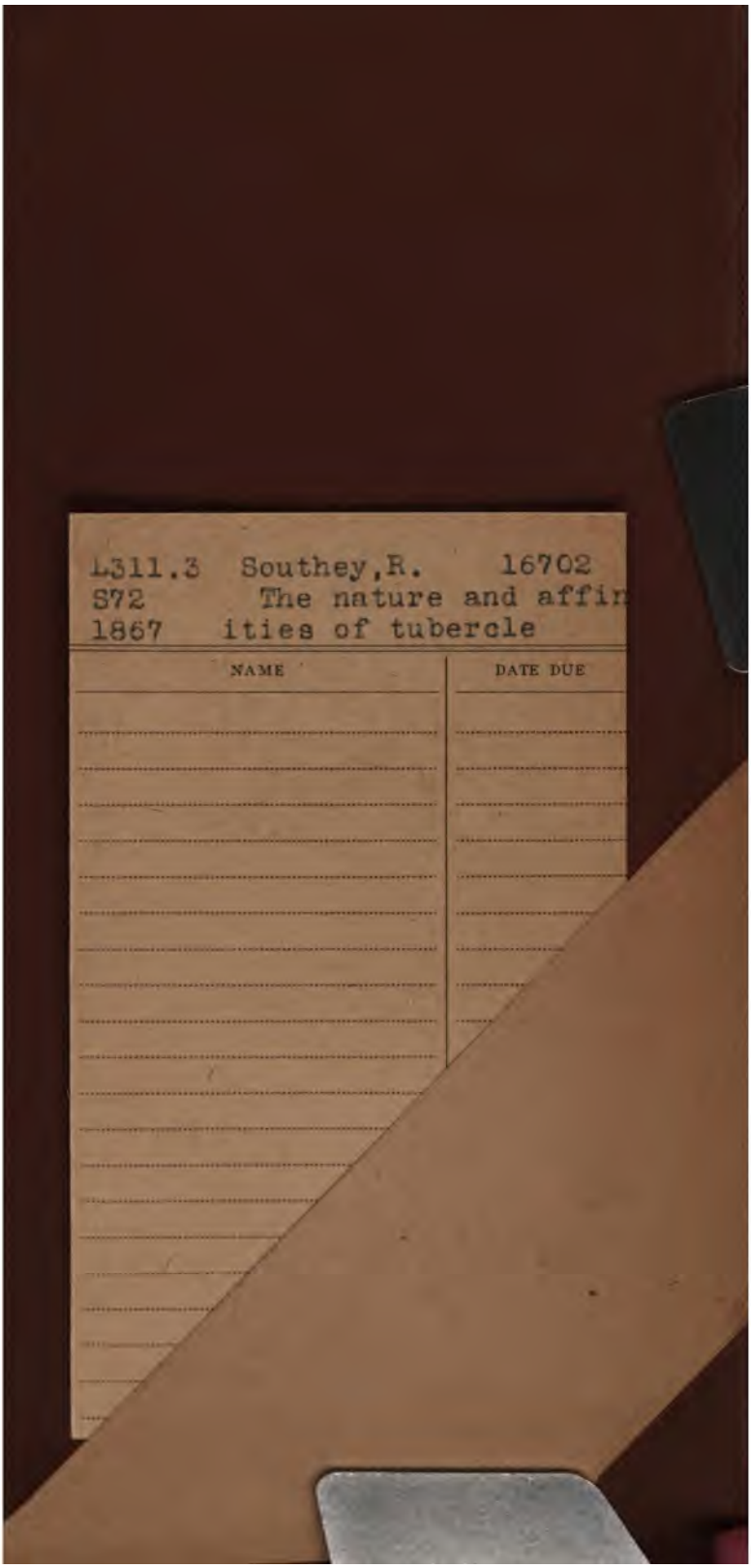
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